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REPORTS

ON

PUBLIC HEALTH AND MEDICAL SUBJECTS.

No. 7.

REPORT

ON THE CAUSATION OF FŒTAL DEATH.

MINISTRY OF HEALTH.

LONDON:
PUBLISHED UNDER THE AUTHORITY OF HIS MAJESTY'S STATIONERY OFFICE.

1922.

Price 10*s. Net*

MINISTRY OF

THE CAUSATION OF FŒTAL DEATH.

Report of an Investigation into the Factors which determined Death in a Sample of Three Hundred Fœtuses of Viable Age.

By

EARDLEY HOLLAND, M.D., F.R.C.S., F.R.C.P.,

Surgeon at the City of London Maternity Hospital, Assistant Obstetrical Physician at the London Hospital.

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REPORT ON THE CAUSATION OF FŒTAL DEATH.

To the Right Hon. Sir ALFRED MOND, Bart., M.P.

SIR, 24th February 1922.

1. I HAVE the honour to submit a special report on the Causation of Fœtal Death.

2. The investigation which forms the subject of this comprehensive and valuable report was commenced under the auspices of the Local Government Board in March 1914 by Dr. Eardley Holland and the late Dr. P. B. Ridge. Owing to the war and to the death of Dr. Ridge and for other reasons delay has occurred in the completion and publication of the report. The report represents an immense amount of careful pathological work in a neglected but very important field of research. It is based upon the examination of 300 fœtuses, a number small indeed in proportion to the number annually still-born in London, but for the present purpose a fairly representative sample, and the provisional conclusions drawn therefore can hardly fail to modify in certain directions the practice and teaching in this important branch of preventive medicine.

3. Dr. Eardley Holland's report is a document of considerable importance, not only because of the valuable research which it describes, but because it should serve to stimulate other investigators to further work in this direction, and it should throw light on the grave issue which is raised when we compare, for any district, the birth rate, the maternal and infant death rates and the fœtal death rate —all of them fundamental data in determining the health of a people. As Dr. Holland points out, our knowledge of fœtal and placental pathology is extremely limited, while the interpretation of conditions observed is often rendered difficult because of our ignorance of the normal histology. Thus an exceptionally promising field of inquiry lies before us.

4. The importance of an exploration into the cause of still-births is obvious. There is an annual loss to the child population from this cause which is much to be deplored and which is largely preventable. With this is associated the loss of strength and energy which has been expended to no purpose by the mothers of these children during their pregnancy and the possible physical injury to the mother from a labour which results in the death of the child. An examination into the causes of fœtal death is bound to add to our knowledge of conditions likely to prove injurious or fatal, and greater knowledge provides us with more effective weapons for preventing death or injury. The high infant death rate during the first few days and weeks of life, which has been little affected by the measures which have proved so successful in reducing the mortality rate of the first year of life as a whole, is doubtless due in part to causes which, in some cases, result in

fœtal death, and we may reasonably hope that a reduction of still-births would be associated with a lowered infant mortality. Here are four substantial grounds for justifying full and further research in this field.

5. Dr. Holland's investigation was concerned with cases obtained from various types of institution and from different districts and care was taken that they should not be in any way selected or differentiated. They may, therefore, be regarded as representative of still-births in general. The numbers are small, and conclusions drawn from the results of the inquiry must consequently be accepted with caution and reserve, but the findings are both arresting and stimulating, and if confirmed by further research and experience may exercise a considerable influence on the practice of obstetrics. Fœtal death may occur at three periods: (1) *ante-natal*, when the fœtus is usually macerated; (2) *intra-natal*, that is, during labour; or (3) *post-natal*, when the fœtus is born with its heart beating, but pulmonary respiration is never established and the fœtus dies without having attained the dignity of a "child." The actual cause of death is often difficult to determine, as there is usually more than one contributing factor, but the deaths have been classified as accurately as possible according to the "primary causes." These are divided into (*a*) maternal states, such as syphilis, eclampsia; (*b*) complications of labour, such as placenta prævia, contracted pelvis; (*c*) placental states, such as retro-placental hæmatoma; and (*d*) fœtal states, such as prematurity, deformities.

6. Among the actual causes of death reference may be made to the two conditions which are considered to be of special interest, namely, syphilis and excessive cranial stress, the first because of its low incidence and the second because of its unexpected importance.

Syphilis.

7. It will be remembered that the Royal Commission on Venereal Diseases was unable to obtain all the information which it desired on the question as to the relationship of syphilis to fœtal death and, therefore, had to rely on indirect evidence. The Commissioners were thus led to give considerable prominence to syphilis as a cause of "still-birth," although they were unable to furnish exact data. Indeed, they felt justified in believing the still-birth rate might be accepted provisionally as an index of the prevalence of syphilis in general. It is, therefore, a matter of particular interest that Dr. Holland has found a relatively small percentage of deaths which he could attribute to syphilis. The examinations made with a view to discovering manifestations of syphilis were thorough, precise and careful, and the co-operation of Dr. Hubert Turnbull was of great value in this connection. Yet among the 300 fœtuses there were only 42 cases of proven syphilis and 6 of "probable" syphilis, or 16 per cent. in all, in addition to 14 cases of "possible" syphilis where there was no

evidence of disease in fœtus or placenta. It is interesting to note that these important findings are in close accord with the results obtained by Dr. Whitridge Williams in Baltimore in 1915, and by Dr. Francis J. Browne of Edinburgh in 1920.

Excessive Cranial Stress.

8. The observations in regard to excessive cranial stress, and their bearing upon the use of forceps and the management of breech deliveries are most valuable and suggestive. Cranial stress is "a compound compressive stress which may be roughly regarded as consisting of two elements: (1) a general compression of the whole head, and (2) a simple longitudinal compression by opposite forces acting at the ends of the long diameter of the pelvis." Dr. Holland discusses in detail the effects of cranial stress on the brain and its supporting membranes and suggests interesting theories as to the effect of "moulding" upon the head and upon the septa of the dura mater while the process is going on. Among 167 fresh fœtuses the tentorium cerebelli was found torn in 81 (48 per cent.), and this was associated with tearing of the falx cerebri in 5 cases and sub-dural hæmorrhage in all but 6. Of these 81 cases, 46 were delivered by the head and 35 by the breech. Forceps were used for 25 of the vertex presentations, the reasons being contracted pelvis, placenta prævia, prolapsed cord, and prolonged second stage; in some of these considerable force was probably unavoidable and in such cases tearing of the tentorium is readily accounted for. Where, however, forceps are applied merely for a prolonged second stage, the occurrence of such injury invites criticism, and Dr. Holland considers that though the use of forceps may be responsible for the saving of many fœtuses, it is also responsible for the unnecessary death or injury of many others.

9. The 35 breech deliveries represent 75 per cent. of the total number of breech deliveries examined, an extremely significant figure. The frequency with which the tentorium is torn during a normal breech delivery has led Dr. Holland to the opinion that the practice of hastening the delivery of the after coming head is responsible for the death of many healthy fœtuses, on account of the excessive cranial stress set up by the forcible passage of the head through a vulval orifice insufficiently dilated and prepared—and he urges the need for a revision of obstetrical teaching in this respect.

Prevention.

10. "The prevention of fœtal death," says Dr. Holland, "is one of the chief aims of the preventive side of obstetrics. But before the fœtal death rate can be diminished its causes must be found." Summing up the causes of death in his sample of 300 fœtuses, he finds 16 per cent. due to syphilis, 10 per cent. to toxæmia of pregnancy, 51 per cent. to complications of labour, including antepartum hæmorrhage, 2 per cent. to chronic renal and other maternal diseases, 6 per cent. to relative

placental insufficiency and retroplacental clot, 5 per cent. to fœtal deformity, and lastly some 10 per cent. in which the cause was undetermined.

11. From this it appears that more fœtuses were killed by the complications of labour than died during pregnancy from maternal or placental disease, a point of great practical importance in its bearing on the teaching of obstetrics and the management of labour. In a rough estimate of the number of fœtuses which in his view died preventable deaths, Dr. Holland states that 52 per cent. or more than half of these children might have been saved, 20 per cent. by ante-natal treatment (this including 16 per cent. syphilitic fœtuses), 20 per cent. by improved technique in treating the complications of labour, and 12 per cent. by a combination of improved ante-natal and intra-natal methods of treatment.

12. The two chief means of prevention of still births suggested by this report are (1) better ante-natal care and (2) an improved standard of midwifery practice to be attained chiefly through the better education of the medical student, but also of the midwife and maternity nurse. Ante-natal hygiene is being slowly but surely developed, partly through ante-natal centres under local authorities at which the expectant mother can obtain advice, partly through ante-natal clinics in connection with maternity homes and hospitals and partly through the midwives and medical practitioners. Its importance is not fully realised by the general public, nor even by doctors and midwives. Yet ante-natal examination and treatment would undoubtedly save a large number of still-births. The present report clearly illustrates the importance of skilled supervision during pregnancy, for the benefit of the child no less than the mother; such supervision can only become effective when the mothers themselves appreciate the necessity of it and are prepared to make use of the facilities provided. Thus educating the mothers to look for ante-natal care is no less important than securing opportunities for obtaining it.

13. The education of the medical student in obstetrics and gynecology still leaves much to be desired, though advances have been made in recent years. It is not so much more time that is needed for this important subject as a more effective use of the time available, better teaching, fuller use of existing opportunities, and improved facilities for the observation of both normal and abnormal cases. The insistent demands of other branches of medicine upon the time of the medical student should not prevent this supremely important section of preventive and practical medicine from receiving an adequate amount of attention. (*a*) Reorganisation of the methods of clinical instruction is probably the reform most urgently called for, at any rate in some of our medical schools. (*b*) Senior teachers are usually not sufficiently concerned with direct practical instruction in the fundamentals of obstetrics, yet a vital subject like midwifery

should not be left in the hands of junior members of the staff. (*c*) Facilities for ward teaching in normal and abnormal midwifery need extension. (*d*) Students should not undertake domiciliary practice alone until they are able to assume reasonable responsibility, not only because such practice may involve risk to mother and child, but also because admirable opportunities of practical teaching are thus lost.

14. Improvements have been made in the training of midwives, but in Dr. Holland's view "the most urgent reform of all, the improved training of doctors, has yet to come; fœtal mortality is not likely appreciably to fall until their standard of midwifery skill is raised." "Ante-natal methods are," he says, "the strategy, intra-natal methods the tactics, of obstetrics. There is, I believe, a tendency nowadays to exalt the strategy at the expense of the tactics. How wrong this unequal conception of midwifery practice is, becomes at once apparent when we realise that when treatment is indicated, as the result of ante-natal examination, such treatment usually consists in the application of intra-natal methods. The only purely ante-natal treatment consists in the treatment of syphilis and of appropriate cases of toxæmia of pregnancy. Otherwise, treatment is by intra-natal methods, such as the induction of premature labour, Cæsarean section, or the careful management of labour. Though ante-natal work is of immense importance, yet the importance of the intra-natal period and the necessity of getting a better standard of midwifery practice must be recognised much more than it is at present." Thus development is needed in ante-natal supervision and improvement in the practical application of the principles of midwifery by all concerned.

I have the honour to be, Sir,
Your obedient Servant,
GEORGE NEWMAN.

Ministry of Health,
Whitehall,
February, 1922.

SECTION I.

Scope and Method of Enquiry.

(1) Introduction.
(2) Sources of the Material.
(3) Methods of Investigation.
(4) Definition of Terms.

I.—(1) Introduction.

It is a curious fact that, until a comparatively recent date, and notwithstanding the brilliant lead given by Ballantyne of Edinburgh, the subject of fœtal death, popularly known as "still-birth," has attracted little attention from obstetricians and pathologists. That the pathologist should have neglected this little-explored, but extremely attractive, field of research is to be explained partly on the supposition that the problems of the dead who have lived are more arresting than the problems of the dead who, legally, have not enjoyed this advantage, and partly by the fact that he has not gained from his obstetrical colleagues the impression that dead fœtuses are pathological material worthy of routine investigation. Neglected by the clinician, the dead fœtus has in consequence been neglected by the pathologist. That the subject has been neglected by the modern obstetrician is, I believe, explained by the fact that the modern obstetrician-gynæcologist has become so enamoured of gynæcological surgery that he is taking less and less interest in the fundamentals of this more arduous, less brilliant, but more important branch of his art. In no other branch of medicine is the standard of teaching so inadequate; it is common knowledge that the student is taught merely enough to achieve an examination which makes little or no demand on his practical knowledge, and passes into practice poorly equipped with skill in the branch which is, perhaps, the most important of all for the welfare of his patients and the good of his country. Obstetrics is the Cinderella of medicine, and is even now beginning to ascend into a proper position amongst the sister branches. This fortunate turn of events is due to the realisation, tardy enough, that obstetrics is one of the greatest of all branches of preventive medicine. The prevention of fœtal death is one of the chief aims of the preventive side of obstetrics. But before the fœtal death-rate can be diminished, its causes must be found, and the object of this investigation has been to draw what conclusions are possible from the study of a comparatively small sample of 300 cases.

I.—(2) Sources of Material.

Considering the number of dead fœtuses of viable age born annually in London, 300 cases seems a comparatively small number, but, nevertheless, their collection was no easy task and required a good deal of organisation. It was necessary that the sample should be a mixed one, representative of several districts and of different types of hospital practice. It is known, for example, that the incidence of syphilis is higher in some districts than in others and that, in hospitals, in-patients yield a higher proportion of fœtuses killed as the result of the severer complications of labour than do out-patients, owing to the frequency with which such cases are transported into hospitals.

The collection of a sample fulfilling these requirements would have been impossible without the help of members of the staffs of several general and lying-in hospitals and poor-law infirmaries; to their generous co-operation I am deeply indebted, for almost without exception, they readily granted me the use of their material. I am especially grateful to the Matrons and Ward-Sisters, who showed no little enthusiasm for the objects of the work, and who readily undertook the duty of immediately notifying me of the cases as they occurred.

The sources of material were the following :—

	Cases.
City of London Maternity Hospital	102
London Hospital	69
General Lying-in Hospital	34
St. Thomas' Hospital	16
St. Bartholomew's Hospital	15
Clapham Maternity Hospital	11
Fulham Infirmary	11
King's College Hospital	8
Middlesex Hospital	8
East End Mothers' Home	8
St. Pancras Infirmary	6
Bermondsey Infirmary	3
Kensington Infirmary	2
Willesden Infirmary	2
Charing Cross Hospital	2
Private Patients	3

I.—(3) Method of Investigation.

Each case was immediately notified to me by printed post-card or telephone; one of my laboratory attendants then fetched the fœtus and placenta, which were placed in a cool chamber and examined as soon as possible. In a day or two I visited the mother, took a complete clinical and obstetrical history, a sample of blood for the Wassermann test and, where possible, a catheter specimen of urine. For her physical condition, I relied

on the hospital case-sheet and on the account of the medical officer in charge.*

The plan of investigation falls under four heads :—(1) The Mother, (2) The Father, (3) The Fœtus, (4) The Placenta, and I used printed blanks, here reproduced, with spaces for the necessary details.

(1) *The Mother.*—The points noted are best seen by examining the blank, and the most important are those dealing with the "Recent Labour"; these details were, humanly speaking, always reliable and were obtained either from the more or less carefully kept hospital case-sheet or from a personal interview with the doctor or midwife who conducted the labour. Under the heading of "Recent Pregnancy," the information I gained about the state of health preceding and during pregnancy was, in the majority of cases, worthless, since it depended entirely on the mother's own statement, or on those of her friends, unless, as was unusual, she was under medical supervision during pregnancy. I had hoped to gain some useful information from enquiring into the economic condition of the family but was disappointed. I soon came to the conclusion that the figures would be too small for statistical purposes, quite apart from the fact that bad economic conditions, except for their association with lack of medical supervision, seemed to have no correlation with the health of the fœtus or the manner of fœtal death. Under the heading of "History of Former Pregnancies," I was at particular pains to ensure accuracy; with tactful and sympathetic questioning, I was able to obtain a reliable account of the number, dates, duration and mode of termination of former pregnancies.

(2) *The Father.*—As originally planned, the investigation was to include a physical examination of the father, a Wassermann test, his health-history and an enquiry into his economic condition and employment. But he was found to be a most elusive person; sometimes he was anxious to co-operate and, if his work allowed, which was seldom, might be persuaded to come for examination. I have had to leave him out altogether, and perhaps important results have been thereby lost; but I am not inclined to think that the health of the father has much influence on fœtal death, except indirectly through the mother—as, for instance, in syphilis.

* It will be noted that though the number of cases is 300, the case-numbers run up to No. 422. This discrepancy is due to the fact that 122 fœtuses were discarded as being useless for the purposes of the investigation; they were all numbered as received in the Laboratory, and the original numbers have been adhered to. The chief reason for discarding them was because they did not reach the standard of viable age (*i.e.*, they were abortions); sometimes the reason was that the fœtus was decomposed, or that I could not get a reliable clinical history, or examine the mother.

(3) *The Fœtus.*—The fœtuses were weighed, measured and subjected to a complete post mortem examination; cultures, aërobic and anaërobic, were taken from the heart-blood, but no post mortem Wassermann test was performed. Except in cases of advanced maceration of the fœtus, microscopical sections of the chief organs were prepared; in advanced maceration the histological details are so much obliterated by autolysis of the cells that sections are not worth cutting. In every fœtus pieces of the liver, spleen, lung, kidney and suprarenal capsule were prepared by Levaditi's method and examined for the presence of *spirochæta pallida;* in the last 200 fœtuses the direct method, of dark background illumination, was also employed. Whenever enough urine could be obtained from the bladder, it was examined for albumen, sugar, acetone and deposit.

The results were recorded on the blank forms here reproduced.

(4) *The Placenta.*—After removal of the membranes and umbilical cord, the placenta was weighed, measured and examined macroscopically for old blood clots, depressions on the maternal surface, infarcts and other abnormalities. A more detailed examination was reserved until after the placenta had remained for a fortnight in a $2\frac{1}{2}$ per cent. solution of formalin. This preliminary fixation and hardening is of great importance for the clear appreciation of changes, especially of red infarcts, in the substance of the placenta. The placenta was then completely sliced through with successive sweeps of a large sharp knife at intervals of half an inch; its whole substance was thus examined for white and red infarcts and for hæmorrhages. Large sections were prepared for microscopical examination.

The umbilical cord was examined for abnormalities of the vessels, ante mortem thrombosis and other errors.

No post mortem examination of a fœtus can be regarded as complete unless accompanied by the examination of the placenta and cord.

No. *Name*

THE MOTHER.

Date Source
Name Age When married
Address

RECENT PREGNANCY.

Result Date of last period
State of health preceding
General health during
Special symptoms during (*i.e.*, swelling of hands or feet, excessive sickness, headaches, fits, hæmorrhage, &c.).
Economic conditions during
If under supervision during

RECENT LABOUR.

Date

Special events or symptoms preceding

Character of labour and complications (*i.e.*, presentation, obstruction, hæmorrhage, forceps, version, &c.).

If child born in absence of skilled assistance

Evidence of abortifacients

Additional Points.

HISTORY OF FORMER PREGNANCIES.

No.	Date of termination.	Duration.	Character of pregnancy.	Character of Labour.	Sex.	Result to Child.	Subsequent Fate of Child.

Additional Points.

PHYSICAL EXAMINATION.

Wassermann reaction Date Result

Date Result

Heart Abdominal organs

Lungs Pelvic organs

Blood Urine

Nervous system

Additional Points.

History of Former Illnesses.

No. *Name*

THE FŒTUS.

Sex Weight Length

POST MORTEM EXAMINATION (Macro. and Microscopic).

External appearance

Brain	Wt.	S.P.*
Thymus	Wt.	S.P.
Thyroid	Wt.	S.P.
Lungs R.	Wt.	S.P.
L.	Wt.	S.P.

* S.P. = Spirochæta pallida.

THE FŒTUS—*continued.*

Heart and Blood-vessels Wt. S.P.

Liver Wt. S.P.

Spleen Wt. S.P.

Pancreas Wt. S.P.

Suprarenals R. Wt. S.P.

L. Wt. S.P.

Kidneys R. Wt. S.P.

L. Wt. S.P.

Generative organs

Bladder

Epiphyses and centres of ossification

Additional Points.

No. *Name*

PLACENTA.

Weight

General appearance

Microscopical examination

Umbilical Cord. length
abnormalities

No. *Name*

THE FATHER.

Age Usual employment

Wages and special conditions of employment during, and immediately preceding, Wife's present pregnancy

Habits

Physical Examination.

Wassermann reaction Date

Heart Abdominal organs

Lungs Urine

Nervous system General physique and appearance

Additional Points.

Results of a former marriage.

I.—(4) Definition of Terms.

Before proceeding further I wish to define a few terms as used in this Report.

Fœtus.—The word "fœtus," so freely scattered throughout obstetrical writing and speech, has suffered much from lack of definition, and clear thinking has suffered in proportion. In any text-book it is made freely interchangeable with such words as infant, child and baby. The period of fœtal existence has a beginning and an end. It may be said to begin at the end of the embryonic period; it is not easy to state when an embryo becomes a fœtus, nor is it of practical importance to define clearly, even if it were possible, the beginning of fœtal existence. But it is of extreme importance to define clearly the end of fœtal existence, and it is not difficult. A fœtus becomes an infant, child or baby as soon as it has breathed or, more precisely, as soon as its existence depends on the oxygen obtained from the lungs and not on that obtained from the placenta or, as is possible for some minutes after birth, on the reserve of oxygen contained in its tissues. The transition from fœtus to infant is a metamorphosis, which is determined by the establishment of pulmonary respiration.

Fœtal Death.—The fœtus dies when its heart ceases to beat. It is of the utmost importance to distinguish the time at which fœtal death occurred. This, indeed, may be said to be the prime fact in an enquiry into the death of any fœtus. Fœtal existence, in relation to its environment, may be said to pass through three stages: (1) in the quiescent uterus—a long stage; (2) in the active uterus, during labour, a relatively short stage; (3) after birth, a transient but variable stage, before the establishment of pulmonary respiration announces that fœtal existence has ended. This stage of post-natal fœtal life deserves, in clinical obstetrics, more notice than it has hitherto received; around it has gathered the confusion and controversy about the expressions "still-birth" and "dead-birth." In most cases, this stage is a transient one and pulmonary respiration is established almost simultaneously with birth. In other cases this stage may be more prolonged; and the failure to recognise that it may be a normal abnormality has been responsible for the death of many a fœtus, erroneously regarded as a case of "white asphyxia" and assaulted with misdirected and vigorous attempts at resuscitation. If this stage of post-natal existence is prolonged, the fœtus is able to maintain its life for a considerable time by the store of reserve oxygen in its tissues. The example which will immediately come to mind is the case of the apnœic fœtus after the use during labour of scopolamine-morphine anæsthesia. Such a fœtus may remain apnœic for as long as 20 minutes and may yet spontaneously breathe and be none the worse for the experience. Another example is the fœtus delivered by Cæsarean section; and another, the fœtus

delivered in cases where there has been considerable cranial compression during labour, as in some cases of forceps and breech delivery.

Fœtal death may occur at any of these stages :—

(1) Death before labour—ante-natal fœtal death. These fœtuses are usually macerated; but if death occurred shortly before labour, the fœtus would not be macerated.

(2) Death during labour—intra-natal fœtal death. These fœtuses are usually in a fresh state, but if death occur in the early stages of a very long labour, the fœtus may be born in a state of early maceration.

(3) Death after labour—post-natal fœtal death. The fœtus is born alive, *i.e.*, with its heart beating, but pulmonary respiration is never established, and the fœtus dies without having attained the dignity of a "child." Nearly all these fœtuses are found, on post mortem examination, to have cerebral hæmorrhage; they are usually called cases of "white asphyxia" which could not be resuscitated. If a "Fœtal-Death" or "Still-Birth" Registration Act comes into force, it will yield no information of real value unless the certificate contains a statement as to the time of death, so far as it lies in the power of the observer to decide. To attain this the usual standard of clinical observation of the fœtus during pregnancy and labour, and after birth, will have to be improved.

I should like to see the terms "still-birth" and "dead-birth" deleted from obstetrical nomenclature. They are commonly held to be synonymous, have given rise to endless confusion, and have no exact scientific meaning. Ballantyne has fully discussed the matter in a paper he published in the *Journal of Obstetrics and Gynæcology of the British Empire* for March 1914. He proposes to use the term "dead-birth" for a fœtus born without any signs of life (*i.e.*, without heart-beat or movement), and to restrict the term "still-birth" to a fœtus which, at birth, exhibits signs of intra-uterine existence (*i.e.*, heart-beat and movement) but which dies without having attained the sign of independent existence (*i.e.*, pulmonary respiration). This is a notable advance in clearness of thought.

For "still-birth" and "dead-birth" I would substitute the term "fœtal death" (or "fœtal death-birth"); instead of a certificate of still-birth I would have a certificate of "fœtal death," differentiated into the three varieties—ante-natal, intra-natal, and post-natal. These expressions will be clear and simple to those who keep clearly in mind the definitions of fœtus and fœtal death, and who understand the physiology of fœtal existence. They may not, at first sight, appeal to members of the legal profession, or to the average midwife; but the progress of obstetrics does not lie with them, nor is there any reason why we should accommodate our obstetrical nomenclature to their standard of obstetrical knowledge.

Viability.—For the purposes of this investigation all fœtuses were held to have been viable whose length, measured from the crown of the head to the heel, equalled or exceeded 35 cms.; or whose period of intra-uterine life, as calculated from the date of the mother's last menstrual period, equalled or exceeded 28 weeks.

SECTION II.

Method of Classification.

(1) Classification of the Causes of Fœtal Death: Distinction between Primary Causes and Consequent Factors.

(2) Table of Primary Causes (Table I.).

II.—(1) The Classification of Causes of Fœtal Death: Distinction between Primary Causes and Consequent Factors.

It is not an easy matter to decide how best to classify the causes of fœtal death. If we try to think the matter out, from concrete examples, we shall find ourselves lost in a bewildering maze of alternative classifications such as—primary and secondary, direct and indirect, predisposing and exciting, maternal, fœtal and placental—and so on. Each of these we shall probably find has its advantages and yet they do not satisfy all cases. As a matter of fact, no classification fulfills every requirement. Furthermore, again in the light of concrete examples, it is not always possible to state what is exactly the cause of death; for in many cases there is a large choice of possible causes. Illustrative examples will best explain these considerations.

In a case of placenta prævia, the following sequence may be found :—

> Placenta prævia—separation of the placenta and hæmorrhage—prolapsed cord—version and delivery—fœtal cranial injury and cerebral hæmorrhage.

What is the cause of fœtal death here? There is a large choice—fœtal asphyxia from separation of the placenta; fœtal anæmia from rupture of placental umbilical branches; fœtal asphyxia from pressure on the cord; fœtal cerebral hæmorrhage.

Or, in a case of contracted pelvis :—

> Contracted pelvis—transverse presentation—version—delivery by the breech—fœtal cerebral hæmorrhage.

What is the cause here? The contracted pelvis, the faulty presentation, or the fœtal injury? The fœtal injury is of course the determining cause, but is it the primary? In both these examples, still another cause might be cited for fœtal death—faulty obstetrical treatment.

Or, the following case (No. 225 in the series) :—

> Slight albuminuria of pregnancy—excessive white infarction of the placenta—death of the fœtus an hour before delivery.

Here the direct or exciting cause is, no doubt relative placental insufficiency. But which is the primary cause? That is to say, was the albuminuria secondary to the placental state or was the placental state secondary to the albuminuria? In other words, was the cause primarily maternal or primarily placental?

Karl Pearson's remark about cause comes in very aptly here. "No phenomenon or stage in a sequence has only one cause; all antecedent stages are successive causes and, as science has no reason to infer a first cause, the sequence of causes can be carried back to the limit of knowledge" (*Grammar of Science*," p. 130). It would be a mistake, in the present enquiry, to attempt to go too far back in the sequence of causes; it would involve the splitting of hairs and the assumption, in many instances, of the truth of aetiological theories which are still far from settled. As the primary aim of this investigation is to help forward the prevention of fœtal death, I shall adopt a simple and practical classification. As the "primary cause," I shall take the gross clinical condition, when there is one. For example, in the examples cited above, placenta prævia and contracted pelvis would be the "primary cause"; prolapse of the cord, delivery by version or forceps resulting in cerebral hæmorrhage would be "consequent factors" (or "secondary causes"), since they are secondary to the primary clinical condition. When there is no gross clinical condition, the pathological state of fœtus or placenta will have to be taken as the "primary cause": such as "retroplacental hæmatoma," or "excessive white infarction," leading to relative placental insufficiency; or fœtal deformities, such as anencephaly and general œdema.

At the same time, these "primary causes" will be divided, as correctly as possible, into (1) maternal states, (2) complications of labour, (3) placental states, and (4) fœtal states.

Following the primary cause—*consequent* to it—the subsequent events leading to fœtal death may be several. Which particular one proves lethal depends chiefly on the primary cause; but it is often a matter of chance, depending much on the obstetrical treatment of the case and on the accidents of labour. These subsequent events I have termed "*consequent factors*." Evidence of the consequent factors is usually found in post-mortem examination of the fœtus and placenta. This method of classification will best be appreciated from examples.

Placenta prævia is a *primary cause*: the *consequent factors* leading to fœtal death may be: (1) separation of the placenta leading to fœtal anaërosis; (2) prolapse of the cord leading to anaërosis; (3) fœtal hæmorrhage from ruptured placental umbilical branches; (4) cranial injury and cerebral hæmorrhage caused by the method of delivery.

Eclampsia is a *primary cause*; the *consequent factors* may be: (1) toxæmia of the fœtus, as demonstrated by necrosis in

the fœtal organs; (2) retroplacental hæmorrhage causing relative placental insufficiency, discoverable on examination of the placenta; (3) birth injuries.

More than one consequent factor can act in any given case.

The chief consequent factor found on examining fœtuses that have died during labour is cranial injury, in the form of tearing of the dura mater septa and subdural cerebral hæmorrhage. Evidence of acute anaërosis is another extremely common post-mortem finding; it usually indicates sudden interference with the utero-placental or fœto-placental circulation, as from retro-placental hæmorrhage or prolapse of the cord.

I feel that the classification I have adopted needs this explanatory apology. It is, I admit, arbitrary and imperfect. I have adopted it, after much thought, because I feel it is practical and, more than any other, leads the eye at once to that stage, in the sequence of events leading to fœtal death, which is amenable to preventive treatment.

The following table displays the "primary causes" of death as found, in the case of macerated and fresh fœtuses respectively, amongst the 300 cases of fœtal death with which this investigation deals.

II.—(2) Table of "Primary Causes."

Table I.

Primary Causes of Death.

	Mac.	Fresh.
Maternal States:		
Syphilis	39	3
Probable syphilis (not primary cause in 2)	4 (1)	(1)
Albuminuria of Pregnancy	14	7
Eclampsia	1	4
Accidental Hæmorrhage	11	20
Chronic Renal Disease	4	
Other Maternal Diseases	3	1
Complications of Labour:		
Placenta Prævia	4	32
Contracted Pelvis		24
Breech presentation		16
Transverse presentation	2	8
Primary prolapse of Cord		10
Coiling of Cord round Fœtus	1	1
Prolonged Labour in—		
Vertex present.		8
Brow present.		3
Face present.		1
Normal Labour, excessive Cranial Stress		9

Primary Causes of Death—continued.

	Mac.	Fresh.
Placental States :—		
Relative Placental insufficiency—		
Retroplacental Hæmatoma	5	
Intraplacental Hæmorrhage	1	
Excessive red or white Infarcts		2
Abnormally small Placenta	10	
Fœtal States :—		
Deformities	2	8
Postmaturity	1	1
Absence of 1 umbilical and hypogastric Artery	3	
Hydramnios and uniovular Twins	1	
Fœtal Infections	2	
Prematurity		1
Drug Poisoning		1
Cause unknown (including cases of possible Syphilis)	25	7
TOTAL, 300	133	167

SECTION III.

SOME OF THE CHIEF PRIMARY CAUSES OF FŒTAL DEATH, WITH THEIR CONSEQUENT FACTORS—

(1) ALBUMINURIA OF PREGNANCY.

(2) ECLAMPSIA.

(3) PLACENTA PRÆVIA.

(4) CONTRACTED PELVIS.

(5) BREECH PRESENTATION.

(6) RETROPLACENTAL HÆMORRHAGE.

III. (1) ALBUMINURIA OF PREGNANCY.

The number of cases is 21; the fœtus was macerated in 14, and fresh in 7.

Case Numbers.—Macerated: 38, 80, 105, 110, 127, 190, 198, 217, 279, 359, 362, 370, 382 and 403. Fresh: 64, 108, 140, 183, 372, 389 and 398.

These are all cases of the typically toxæmic type; cases of albuminuria due to chronic renal disease or associated with accidental hæmorrhage or retroplacental hæmatoma are not included.

Details of the length and weight of the fœtus, and weights of the fœtal organs and placenta will be found in tables.

Consequent Factors.—If the fœtus is born dead, present routine experience ascribes death to one of four factors :—

(1) The direct action of the maternal poisons.

(2) The associated placental state.

(3) Well- or ill-directed therapeutic obstetrical procedures.

(4) Adventitious states.

In the case of macerated fœtuses the third factor is excluded. In the present series of 14 macerated fœtuses the placenta was not available for examination in cases 80, 105, 279, 362, 382 and 403, and, of the remainder, was not microscopically examined in 359 and 370. There were interstitial placental hæmorrhages in cases 190 and 217, and extensive red infarction in cases 110 and 217. There was an excessive number of white infarcts in cases 127 and 372, but not nearly sufficient to cause death, as judged by the standard of case 225 (*q.v.*). On microscopical examination moderately enlarged avascular villi were found uniformly distributed in the sections in cases 38 and 198. Much enlarged œdematous villi were found in case 190, and no change was noted in sections from cases 110 and 217.

In cases 80 and 370 the maternal Wassermann reaction was positive, but there was no other evidence of syphilis. One case of syphilis (No. 102), had albuminuria of pregnancy, but is not included in the above list, as the primary factor in that case is considered to be syphilis, and the albuminuria of pregnancy a coincidental association.

In these 14 macerated fœtuses, it is not unreasonable to assign death to the placental state in the five cases with red infarctions and intraplacental hæmorrhage, aided by the action of the maternal poisons. In cases where no placental changes are found, maternal poisons must be blamed.

In fresh fœtuses the additional consequent factor of obstetrical treatment plays its part. Of the present series of seven cases, in four (Nos. 64, 183, 372 and 389) there was no interference, nor was anything striking found in the placenta, so death may be ascribed to maternal poisons. Reliable observations about the fœtal heart sounds were only given me in case 389, where they were noted to cease about two hours before delivery.

In three, labour was induced; in two, consequent on the use of a Champetier de Ribe's bag, the cord prolapsed, with results fatal to the fœtus; in the third, the second stage of labour was extremely rapid and the fœtus was found to have bilateral tearing of the tentorium cerebelli, and cerebral hæmorrhage.

Fœtus 108 was noted to be alive shortly before the cord prolapsed; following on this accident, high forceps was applied. Subsequent examination revealed a depressed fracture of a parietal bone, tears in the tentorium, and much cerebral

hæmorrhage. Thus, in order to protect it from death by maternal toxins, this fœtus was killed twice over by well-intentioned obstetrical procedures: (1) the prolapse of the cord, (2) the cranial stress.

III. (2) Eclampsia.

There were five cases of eclampsia (four fresh fœtuses and one macerated).

No. 28.—Fresh premature fœtus delivered by Cæsarean section. The fœtal liver showed large areas of necrosis of cells, and interstitial hæmorrhage; the epithelium of the kidney tubules also was extensively degenerated (but this degeneration of kidney-cells is a change often found in the fœtal kidney apart from toxæmic states). There was no evidence of retroplacental hæmorrhage, but the placenta contained several areas of recent red infarction. The consequent factor leading to fœtal death here was probably toxæmia; it may have been relative placental insufficiency, but there were no signs in the fœtus of acute anaërosis.

No. 48.—Fresh premature fœtus. Signs of acute anaërosis, *i.e.*, many subpleural petechial hæmorrhages, and the same in the thyroid and the visceral pericardium; hæmorrhage between layers of falx cerebri. External surface of liver not quite uniform in colour, but no distinct mottling; the surface was mostly dark red, with slightly paler areas. The cut surface had the same appearance; no interstitial hæmorrhages. Microscopical examination showed many areas of focal necrosis and small interstitial hæmorrhages; almost complete necrosis of all the cellular elements of the kidney—a massive necrosis. Examination of the placenta revealed retroplacental hæmorrhages and much recent red infarction; in two places were depressions on the maternal surface occupied by old blood clot. There was also intraplacental hæmorrhage. There were three large areas of recent red infarction.

The consequent factors causing fœtal death in this case were: (1) relative placental insufficiency, (2) toxæmic necrosis of the liver, kidneys, and other organs.

No. 207.—Moderately premature fresh fœtus. Placenta not examined. Too much postmortem degeneration for a satisfactory histological examination of fœtal organs. Slight subdural hæmorrhage over occipital lobes.

No 286.—Moderately premature fresh fœtus. The liver showed three subcapsular hæmorrhages; histological examination of fœtal organs not made. The placenta contained five small white infarcts and elsewhere consisted almost entirely of large areas of recent red infarction; the term "massive red necrosis" would apply to this placenta.

No. 49.—Macerated premature fœtus. Placenta not examined. This case yields no information.

The consequent factors in fœtal death in these cases were either : (1) relative placental insufficiency from retroplacental hæmorrhage or red infarction; (2) toxæmic necrosis of the liver, kidney and other organs. Both of these may be found together in the same case.

III. (3) Placenta Prævia.

The number of cases is 36; the fœtus was macerated in four, and fresh in 32.

Case Numbers.—Macerated : 22, 170, 180 and 271. Fresh : 17, 19, 82, 103, 116, 136, 142, 165, 166, 187, 203, 208, 236, 255, 261, 263, 265, 269, 295, 296, 302, 326, 338, 343, 344, 353, 360, 373, 374, 379, 383 and 404.

There are five other cases where the placenta was prævia, but in which it was not taken as the primary factor. Two of them were cases of fœtal syphilis (141 and 149); one was a case of general œdema of the fœtus (391), and two were cases of relative placental insufficiency due to small placenta (278 and 322). In the two last there was no antepartum hæmorrhage, but the evidence rested on the examination of the placenta and membranes. It is interesting to note the effect of fœtal death on the extent of antepartum hæmorrhage in cases of placenta prævia. Where the fœtus has been dead for a long time before the onset of labour, there is little or no hæmorrhage, no doubt because of thrombosis of the blood in the decidual vessels. Where, on the other hand, the fœtus dies only shortly before the onset of labour, the hæmorrhage is as severe as usual. In cases 278 and 322 the fœtus was in an advanced stage of maceration; so was the fœtus in case 141, and the hæmorrhage was insignificant. In case 149 the maceration was slight, and the hæmorrhage, which began only twelve hours before delivery, was severe.

Where placenta prævia is the primary factor, the consequent factors leading to fœtal death are :—

(1) Fœtal anaërosis from extensive separation of the placenta.

(2) Fœtal anaërosis secondary to severe maternal anæmia.

(3) From complications of labour commonly associated with placenta prævia, such as prolapse of the cord and transverse presentation.

(4) Fœtal anæmia from rupture of placental umbilical branches or of *vasa prævia*.

(5) From well- or ill-directed therapeutic obstetrical procedures.

When fœtal death is due to placenta prævia the fœtus is seldom macerated, since it is either born soon after its death or, more commonly, has been killed by the process of delivery.

In the four cases of macerated fœtus in this series, severe antepartum hæmorrhage had occurred three or more days before delivery. In such cases, if other known causes of death can be excluded, we may reasonably assume that the fœtuses died from the effects of placental separation at the time of the first hæmorrhage and were retained for a length of time sufficient for maceration.

When the fœtus is recently dead, even the most exact clinical and post mortem observations may fail to determine what killed it. I have divided the consequent factors into five classes, and the difficulty lies chiefly in the fact that these factors more often act in combination or in sequence than singly. The great practical importance of the enquiry lies in finding out whether the fœtus was killed by the natural consequences of placenta prævia, or by obstetrical treatment. Placenta prævia is a dangerous maternal complication, and the life or health of the mother must not be jeopardised for the sake of such a doubtful quantity as the unborn fœtus. The best methods of treatment have been worked out by many well-known observers, and those methods which give the lowest maternal and fœtal mortality are now well standardized. There is no doubt that the fate of the fœtus in cases of placenta prævia is largely dependent on the knowledge, skill and opportunities of the obstetrical attendant. There must, of course, always be a high fœtal mortality, but it can be considerably lowered by raising the standard of obstetrical practice.

Obstetrical procedures, apart from the natural consequences of placenta prævia, may kill the fœtus (1) by causing wide separation of the placenta or by tearing through umbilical vessels; (2) by causing too rapid delivery of the head by forceps or leg-traction, resulting in excessive cranial stress; (3) by bringing about prolapse of the cord—a common consequence of internal version.

The details of these cases will be found in Table X. (A) and (B), and they will repay study. The clinical observations have not been sufficiently accurate to enable me to draw any but the broadest conclusions.

Auscultation of the fœtal heart sounds helps to decide whether the fœtus died before or after the employment of obstetrical treatment: if the cord was found pulsating at birth, the fœtus probably did not die from cord-pressure or separation of the placenta.

On post mortem examination, signs of acute anaërosis point to cord-pressure or separation of the placenta as the most likely mode of death: remarkable anæmia of the fœtal tissues points to fœtal hæmorrhage from the umbilical vessels: tears of the tentorium cerebelli point to cranial stress from too rapid delivery. I do not, for a moment, conclude that, if evidence of cranial stress is found, the fœtus has necessarily been killed by cranial stress; for this is just as likely to occur in a dead fœtus as in

a live one. It is precisely here where accurate auscultation of the heart sounds is of service. If evidence of cranial stress is found in a fœtus whose heart was beating before the act of delivery, it is reasonable to conclude that the act of delivery killed it. If, again, evidence of cranial stress is found in a fœtus whose heart was beating at birth, the same conclusion is justified.

In these 32 fresh fœtuses tears of the tentorium cerebelli (with cerebral hæmorrhage in all but three) were found in 16, or 50 per cent.; four of these were delivered by forceps, and 12 by leg-traction after version. How many of these 16 were killed by cranial stress I do not know, for the necessary clinical observations are lacking. Nos. 263 and 265 I know were not, for both these fœtuses were as bloodless as possible. In any case it is a mistake to kill a fœtus twice.

In my opinion, the mistakes commonly made in the treatment of placenta prævia, and which are responsible for so much of the fœtal mortality are :—

(1) Over-treatment.—Some cases, with only slight hæmorrhage, require no treatment beyond rupture of the membranes.

(2) Internal Version.—The indications for this operation are very limited; the cord often prolapses during its performance. Much better is external version, or bipolar version, followed by bringing down a leg.

(3) Too rapid delivery.—This is usually as dangerous for the mother as for the fœtus. I believe it accounts for the majority of the avoidable fœtal deaths. Once hæmorrhage is controlled the delivery of the fœtus should be the slowest possible.

III. (4) Contracted Pelvis.

There are 24 cases of contracted pelvis.

Case Numbers (all fresh fœtuses).—33, 111, 135, 175, 179, 231, 235, 237, 248, 252, 258, 259, 303, 331, 346, 349, 350, 386, 409, 410, 417, 418, 420 and 422.

In all these cases contracted pelvis is undoubtedly the primary factor; the consequent factors are either the complications of labour natural to contracted pelvis or to the obstetrical procedures undertaken to overcome these complications. In these 24 cases the consequent factors may be summarised in 21 as follows :—

	Cases.
Prolonged or obstructed labour, forceps, cranial stress	9
Obstructed labour, craniotomy (ante or post mortem)	7
Obstructed labour, forceps, prolapsed cord, internal version, cranial stress	1
Obstructed labour, internal version, prolapsed cord, cranial stress	1
Impacted transverse presentation, decapitation	1
Transverse presentation, version, cranial stress	1

In one other case (346) of forceps for prolonged labour there was no tearing of the tentorium cerebelli, but death may have been due to cranial stress, during which the tentorium was strained, but not strained to the tearing point.

In the three remaining cases (235, 259 and 409) labour was induced, without success for the fœtus. In one a bag was used, transverse presentation resulted, and the fœtus was delivered by version; the tentorium was torn and there was cerebral hæmorrhage; in another, labour was complicated by coiling of the cord around the fœtus; in the third, death could not be accounted for, either from clinical or post mortem evidence.

Contracted pelvis should very rarely lead to fœtal death, for it should be detected during pregnancy and the fœtus saved by proper obstetrical treatment. Fœtal deaths from this cause will continue *ad infinitum* until women are under proper supervision during pregnancy. Detection of advanced degrees of contraction is so easy as to be almost obvious, even to the most ill-trained observer. But in the vast majority of cases of pelvic contraction the contraction is slight and is only to be detected by measurements and by tests during the last few weeks of pregnancy. Such observations require a degree of skill only to be acquired by careful training and long experience.

Out of these 24 cases, in only five was the mother under supervision during pregnancy. In three of these, premature labour was induced, with results unsuccessful to the fœtus; in the remaining two, the contraction was either undetected, or, if detected, was considered so slight as to give reasonable hope of a successful labour at the full term of pregnancy. These five fœtuses at all events had the advantages of obstetrical science. In the other 19, the mothers were not examined during pregnancy, and the condition was only revealed during labour by the ensuing complications. This is, or should be, inexcusable; still more heart-breaking are those cases where the fœtus was similarly sacrificed in former pregnancies. Thus, out of the nine multiparæ amongst the 21 unsupervised women, in cases 111 and 237 three former fœtuses were killed during labour as the result of contracted pelvis; in cases 349 and 422, two; in cases 171, 331, and 410, one; in the remaining two, cases 252 and 386, former pregnancies terminated successfully, no doubt because the fœtuses were small. These last two cases illustrate the well-known fact that women with slight pelvic contraction can pass through several labours successfully, and only have trouble when the fœtus happens to be larger than usual—an event which generally occurs sooner or later. Multiparæ are, therefore, just as worthy of supervision as primigravidæ.

Case Numbers.—Forceps, cranial stress: 33, 135, 175, 179, 231, 350, 418, 420, 422. Craniotomy: 237, 248, 258, 349, 331 410, 417. Induction: 235, 259, 409. Obstructed labour, forceps, prolapsed cord, version: 303. Obstructed labour, version,

prolapsed cord : 386. Transverse presentation, version, cranial stress : 111. Transverse presentation, impaction : 252.

III. (5) Breech Presentation.

In considering breech presentation as a factor in fœtal death it is of especial importance to keep in mind the distinction between primary and consequent factors. Breech presentation, or, rather, breech labour, was the primary factor in fœtal death in 16 cases, and was a consequent factor in many other cases, in which, for example, the primary factor was antepartum hæmorrhage or contracted pelvis.

I will state at once the remarkable fact that tears of the tentorium cerebelli and cerebral hæmorrhage are almost constantly found in dead fœtuses delivered by the breech; this throws a flood of light on the cause of fœtal death in breech labour and indicates precisely the obstetrical faults in its management.

Of the 168 fresh fœtuses, 47 were delivered by the breech; they may be divided into three classes :—

(1) *Primary uncomplicated breech presentation.*—This class consists of the 16 cases of fœtal death during labour, and in which breech presentation was the primary factor in fœtal death. In none of them was there any complication of pregnancy, or complications of labour (except premature labour), other than those arising directly from the presentation. One other case falls into this class, a case of general œdema of the fœtus (No. 87). The primary factor in death was here the fœtal state and not the breech delivery, so it will not be further considered in this section.

(2) *Primary complicated breech presentation.* — There are two cases in this class : one of accidental hæmorrhage, one of placenta prævia.

(3) *Breech by version.* — There are 28 cases in this class, consisting of 2 cases of accidental hæmorrhage, 14 of placenta prævia, 8 of transverse presentation, and 4 of contracted pelvis.

Class I.

Case Numbers (excluding the case of general œdema).—42, 57, 134, 163, 177, 200, 205, 226, 272, 283, 314, 334, 356, 363, 397, 401.

Details of the maturity of the fœtus will be found in Table I; it will be noted that six mothers were primiparæ and four of the fœtuses (Nos. 42, 163, 200 and 286) were 43 cm. or less in length.

On post mortem examinations of these fœtuses, the striking fact comes out that, in all except two (Nos. 134 and 334) there was found tearing of the tentorium cerebelli and cerebra

hæmorrhage—signs of excessive cranial stress. In case 272 labour was complicated by prolapse of the cord.

The fact that 14 out of 16 fœtuses which died as the result of breech delivery showed, post mortem, signs of excessive cranial stress is of great importance. The causes and effects of cranial stress are fully described in a special section, and here I shall only discuss its special assocation with breech delivery.

Breech labour is accompanied by a mortality rate which varies with the skill of the accoucheur; on the average it is seldom less than 8 or 10 per cent. When we realise *why* breech delivery kills fœtuses, we are in a position to revise our methods and to base them on fact and not, as hitherto, on tradition. I believe that it is entirely wrong to hasten the delivery of the after-coming head; yet this is the usual practice. The customary teaching is that the danger to the fœtus arrives when it has been so far born that the cord is compressed between the fœtus and the birth-canal; hence the rule—when the fœtus has been born as far as the umbilicus, its complete delivery must be hastened lest it die of cord compression. In consequence it is the usual practice to hasten the delivery of the head and to extract it within a few seconds of the birth of the trunk. It is this obstetrical procedure which, in my opinion, so often kills the fœtus, and my opinion is based on the fact that I have nearly always found signs of excessive cranial stress in fœtuses killed during breech delivery.

It is not difficult to imagine the danger of hastening the delivery of the after-coming head; the head is subjected to sudden and severe stress, for it is forcibly dragged through a vulval orifice and past a perineum which are unprepared for its passage—or, at all events, only prepared by having been moderately distended by the passage of the body of the fœtus. Compare this with a vertex presentation, where the passage of the head is only permitted after these maternal parts have been subjected to a preparatory distention lasting one or more hours. The death-rate of breech fœtuses will fall when it is realised that they are killed, not by cord compression, but by excessive cranial stress, and when this knowledge has brought about a corresponding change in our present obstetrical procedures.

There is no doubt that the cord is compressed after the fœtus has been born beyond the umbilicus, but I believe the danger of such compression to the life of the fœtus has been much exaggerated. I do not think the fœtus is any the worse for 10 or 15 minutes of cord-compression. That the fœtus can live for some time on its tissue-reserve of oxygen is a fact that has been especially demonstrated since the use of scopolamine-morphine anæsthesia: such fœtuses are often born in a state of apnœa and remain so for 20 minutes or more, and may yet spontaneously revive and be none the worse for their experience.

A new rule must be adopted for breech delivery. Instead of using forceful haste we must be deliberate and deliver gently

and gradually the after-coming head. We must have more respect for the great fragility of the fœtal head, and remember that the obstacles that arrest its delivery, and that must be gradually overcome, are the rigid vulval ring and perineum.

The little operation of episeiotomy is of the greatest value in protecting the after-coming head from excessive stress in cases where the vulval ring and perineum are rigid and resistant; the cuts should be made when the breech is distending the perineum. The delivery of the head is then made surprisingly easy. It is not enough realised that the chief indication for episeiotomy is a fœtal one—to save the fœtal head from excessive stress; the saving of a perineal tear is of secondary importance. I believe that no fœtus should die from simple breech delivery (excluding prolapse of the cord) if the principles I have laid down are put into practice.

Classes II. and III.

Breech delivery, in these cases, was not the primary factor in fœtal death; but it cannot be doubted that, considering the large number of these fœtuses that had tears of the tentorium cerebelli and cerebral hæmorrhage, it was often the consequent factor.

In Class II. there are two cases: No. 27 was a case of accidental hæmorrhage; No. 296, a case of placenta prævia. In neither was there any tearing of the tentorium cerebelli or cerebral hæmorrhage.

In Class III. there are 28 cases. The version was performed for the following complications :—

For accidental hæmorrhage, Nos. 76, 294.

For placenta prævia, Nos. 136, 142, 165, 187, 208, 263, 295, 302, 343, 344, 374, 379, 383 and 404.

For transverse presentation, Nos. 111, 123, 209, 216, 245, 268, 301 and 393.

For contracted pelvis, Nos. 303 and 386.

For brow presentation, Nos. 305 and 339.

Amongst these fœtuses, 20 had tearing of the tentorium cerebelli and cerebral hæmorrhage. Those which showed no such signs of excessive cranial stress were Nos. 76, 123, 208, 216, 295, 379, 383 and 393.

Taking all these classes together, there are 47 cases of breech delivery, of which 35 fœtuses had tearing of the tentorium cerebelli and cerebral hæmorrhage. Thus, in the present series of dead fœtuses (excluding all macerated fœtuses, of course), approximately 75 per cent. of those delivered by the breech showed signs of excessive cranial stress. This, to me, is a very noteworthy fact.

Taking Class III., of 28 cases, in which breech delivery by version was the obstetrical treatment for grave complications of pregnancy and labour, there is no doubt that a certain number

of fœtuses (unfortunately I cannot state how many) had already been killed, before delivery, by the primary complication; but I cannot doubt that many of these fœtuses escaped death from the primary complication only to be killed by the obstetrical procedure undertaken for its relief. The lesson these cases teach is of great importance, and the remarks I have already made under Class I. apply also to Classes II. and III.

III. (6) Retroplacental Hæmorrhage.

Separation of the normally-situated placenta is always accompanied by retroplacental hæmorrhage. Such hæmorrhage is usually profuse, in which case it is evident clinically and is known by the peculiarly meaningless name of "accidental hæmorrhage"—external or concealed or both, as the case may be. In a smaller number of cases the hæmorrhage is slight and gives no clinical evidence of its occurrence; these cases may be referred to as cases of "retroplacental hæmatoma," and the condition can obviously only be detected by examination of the placenta, the maternal surface of which is seen to be depressed or hollowed out by more or less old blood-clots (Fig. 17). Such cases are commoner than is supposed.

Cases of separation of the normally situated placenta thus fall into two groups :—

(1) Those with wide separation; cases of accidental hæmorrhage—the common type.

(2) Those with very slight separation; cases of retroplacental hæmatoma—the rare type.

It is scarcely necessary to point out that retroplacental clot and depressions in the maternal surface of the placenta are found also in cases of "accidental hæmorrhage." The difference between the two types is merely one of degree—the degree of separation and the degree of hæmorrhage. Indeed, the two are often combined in the same case (Fig. 15). Let me illustrate this point by referring to the fate of the fœtus in these cases. In cases of "accidental hæmorrhage" the fœtus, if it dies, as it usually does, is nearly always born in a fresh state; for it is either quickly expelled by the natural forces of labour, or is extracted by the timely assistance of the obstetrician. In cases of "retroplacental hæmatoma," if the placental separation is wide enough to kill the fœtus, the fœtus is retained in the uterus sufficiently long to become macerated; for the uterine disturbance is not sufficiently great to initiate labour, nor are there any maternal symptoms to call for obstetrical interference. But in some cases of "accidental hæmorrhage" the fœtus is born macerated; there are 11 in this series. In four of these the placenta was found to contain a well-marked depression on its maternal surface, filled with old clot (Fig. 16). The explanation is fairly obvious. There was first slight hæmorrhage; the case was one of "retroplacental

hæmatoma," the fœtus died and was retained. Then there occurred further separation with profuse hæmorrhage; the case became one of "accidental hæmorrhage" and a macerated fœtus was born.

In this series of 300 cases there are 36 cases of fœtal death due to separation of a normally situated placenta; 31 are cases of so-called "accidental hæmorrhage" and 5 of "retroplacental hæmatoma" without clinical signs or symptoms of hæmorrhage. In the 31 cases of "accidental hæmorrhage," 20 of the fœtuses were fresh and 11 macerated; the fœtus was macerated in all the 5 cases of "retroplacental hæmatoma."

Case Numbers. Accidental Hæmorrhage.—Fresh fœtuses: 13, 27, 36, 46, 47, 76, 143, 159, 167, 191, 195, 204, 218, 277, 293, 294, 317, 366, 368 and 390; macerated fœtuses: 26, 44, 133 192, 262, 280, 282, 291, 335, 367, 412. *Retroplacental Hæmatoma.*—11, 197, 228, 287, 357.

It is sometimes clinically impossible to distinguish between cases of slight "accidental hæmorrhage" and cases of placenta prævia where the placenta is out of reach of the examining finger; examination of the placenta and membranes settles the diagnosis. It is quite possible that one or two of the eight cases in which I did not receive the placenta for examination may be of this nature.

The causation of fœtal death in separation of the placenta needs no discussion; all the fresh fœtuses revealed, post mortem, signs of acute anaërosis. There remain two points which are worthy of description, the presence of albumen in the urine and the condition of the placenta.

Catheter specimens only were used to determine the presence of albumen, and I was not able to obtain a specimen in all cases. Of those examined, in some the urine was not obtained until three or four days after labour, by which time albumen may have disappeared. Even so, the results are worth recording.

(A) Cases of accidental hæmorrhage :—

(1) With fresh fœtus. Number of cases: 20; urine examined in 12; albumen present in 8, absent in 4.

(2) With macerated fœtus. Number of cases, 11; urine examined in 8; albumen present in 6, absent in 2.

(B) Cases of retroplacental hæmatoma :—

Urine examined in 4; albumen present in 2, absent in 2.

Case Numbers with Albuminuria—Accidental Hæmorrhage.—Fresh fœtuses: 13, 36, 167, 191, 277, 293, 317, 366. Macerated fœtuses: 26, 133, 192, 335, 367, 412. *Retroplacental Hæmatoma.*—197 and 287.

The amount of albumen varied from a cloud to a considerable amount, and I never failed to find tube-casts.

The placentas were examined particularly for red and white infarcts and for intraplacental hæmorrhage, with the following results :—

(A) Accidental Hæmorrhage.

(1) With fresh fœtus, 20 cases. Placenta available for examination in 13 cases; red infarction found in 8 (Nos. 13, 143, 167, 191, 195, 277, 293, 390), none noted in 5.

(2) With macerated fœtus, 11 cases. Placenta available for examination in 10 cases; red infarction found in 9 (Nos. 26, 44, 133, 192, 280, 282, 291, 335, 412), none noted in 1; excessive white infarction found in 1 (No. 44); depressions on maternal surface, with adherent retroplacental clot, found in 4 (Nos. 26, 133, 335, 412); intraplacental hæmorrhage found in 2 (Nos. 280 and 291).

(B) (3) Cases of Retroplacental Hæmatoma. — All the placentas were available for examination and all showed red infarction; in one (No. 228) there was intraplacental hæmorrhage.

SECTION IV.

Fœtal Syphilis.

(1) Method of Investigation for Fœtal Syphilis.

(2) The Diagnosis of Fœtal Syphilis.

(3) Table II.; The Spirochæte-Positive Fœtuses. Table III.; The Spirochæte-Negative Fœtuses with Positive Maternal Wassermann Reaction or other signs of Syphilis.

(4) "On the Recognition of Congenital, Syphilitic Inflammation of the Long-Bones." Specially written for this Report by Professor Hubert Turnbull, D.M.

One of the most important aims of this investigation was to find out the frequency of fœtal syphilis. Our knowledge on this point is at present unsatisfactory; we have no precise information, for no sufficiently large series of cases in this country has yet been investigated. Since the present sample of 300 fœtuses is a mixed one, the fœtuses having been collected not from one but from several different hospitals and areas, it is fair to assume that the incidence of syphilis it reveals is representative for London; how far it is representative for the country as a whole is a debatable point, but the difference cannot be a great one.

In considering the cause of death in those fœtuses in which evidence of the disease is found, a distinction should be made between two groups of cases : (1) those fœtuses in which syphilis is the actual cause of death ; (2) those in which the actual cause of death is not syphilis but some other disease or injury. Evidence of syphilis may occasionally be found in fœtuses killed, for example, by birth-injury or separation of the placenta. In such cases the cause of death must not be placed under the category of syphilis ; but for an accident of labour, such fœtuses would have been born alive to continue their existence as congenitally syphilitic infants. The distinction between the two groups may appear a fine one, but it is necessary when one is trying to arrive at the exact cause of death in any given case. In the following account of fœtal syphilis all cases have been grouped together, and I have made no distinction beyond drawing attention to those cases in which the actual cause of death was other than syphilis.

IV. (1) Method of Investigation for Fœtal Syphilis.

Every case was investigated for evidence of syphilis. The routine consisted of :—

(1) Examination of the fœtal organs for the spirochæta pallida.

(2) The Wassermann reaction in the mother, and. where possible, in the father.

(3) Weights and measurements of the fœtal organs.

(4) Examination of the ends of the long bones for osteochondritis.

(5) Histological examination of the organs of the fresh fœtuses.

(6) Weight and histological examination of the placenta.

1. *Examination of the Fœtal Organs for the Spirochæta Pallida.*

Levaditi's method was employed in all cases and, in about the last 200, the direct method by dark-background illumination was also used.

The original Levaditi method was adhered to, and the organs examined were the liver, spleen, suprarenal, kidney and lung. Thin pieces of tissue must be taken. They are first placed in a 5 per cent. solution of formalin for 24 hours. They are then placed in distilled water until they sink ; next they are transferred to 90 per cent. alcohol for 24 hours. They are then rapidly washed in distilled water and placed in a 1·5 per cent. solution of silver nitrate (pure) in distilled water, and kept in an incubator

at 37° C. in the dark for five or six days. They are then rapidly washed in distilled water, and are developed, still in the dark, for 24 hours at the room temperature in the following solution :—

Pyrogallic acid - - - -	2 gms.
Formalin - - - - - -	5 cc.
Distilled water - - - - -	100 cc.

After this they are again washed in distilled water, hardened in alcohol, cleared in xylol and embedded in paraffin. Thin sections are cut and mounted for examination. The spirochætes are stained black, connective-tissue fibres dark brown, and protoplasm yellow. A great essential for success is scrupulous cleanliness; all solutions must be made with distilled water, and dishes and bottles must be washed out with distilled water before use. The silvering and development should be carried out in the dark. In the direct method the thinnest possible film of an emulsion of the fresh organ is made between a slide and a very thin cover-slip, and is examined with a $_1{}^1{}_2$-inch oil-immersion objective over a dark back-ground illumination stage. A convenient way to make an emulsion is to scrape with a needle the freshly-cut surface of the organ and to take some of the juice with a fine pipette. A tiny drop only must be used, and the coverslip must be pressed down firmly on the slide. If spirochætes are not found at the first attempt other areas are examined. The organs which lend themselves best to this method are the more deliquescent ones such as the liver and spleen; but where spirochætes are abundant, they are to be found in any organ or part of the body (including the brain and the epiphyseal line of the long bones). The advantages of the method are speed and simplicity. A positive result settles the diagnosis at once and saves the time spent over the long and tedious Levaditi method. If the organ contains spirochætes they are usually quickly found; only in one case was a prolonged search necessary before a single spirochæte was found. If, however, spirochætes are not found by the direct method, it is not safe to exclude them from the organs until these have been examined by the Levaditi method. In one case, spirochætes were missed by the direct method, but were found in Levaditi sections. This was the case of an extremely macerated fœtus which was not received for examination until the fourth day after birth; in the Levaditi sections only a few perfect spirochætes were found, for they had nearly all undergone granular disintegration. In all cases the Levaditi method was employed in addition to the direct method, even where the latter had given a positive result, to learn the relative value of the two methods.

The advantage of the Levaditi method is that the sections form a permanent record; sections, as well as prepared blocks of tissue, can be kept and, in doubtful cases, searched again at a future date for spirochætes, which may possibly have eluded the original search.

2. *The Wassermann Reaction.*

In every case the maternal Wassermann reaction was tested. The blood was usually taken within a week of delivery. It was found so difficult to arrange a meeting with the father, or to get him to attend the laboratory, that the original intention to test his Wassermann reaction was soon given up. The tests in about the first 250 cases were carried out in the bacteriological laboratory of King's College Hospital by the late Dr. P. B. Ridge, and in the later cases in the bacteriological department of the London Hospital by Dr. G. T. Western.

3. *Weights of the Fœtal Organs.*

The ratio of the weights of the important organs to the weight of the fœtus was calculated in all cases and will be found displayed in tables, both for syphilitic and for non-syphilitic fœtuses, at the end of this Report. The alterations in the weight-ratios of syphilitic fœtuses, and the significance of such alterations as evidence of syphilis, will be discussed later. The enlargement of certain fœtal organs, notably the liver and spleen, is common in syphilis, but is not constant. It may be noted at once that such enlargement is not peculiar to syphilis, as it was often found amongst the non-syphilitic fœtuses. Nevertheless, the enlargement of certain organs forms useful evidence of secondary importance which, taken in conjunction with other evidence, helps towards a decision in doubtful cases.

4. *Histology of the Fœtal Organs.*

Owing to certain vicissitudes occurring during the course of this investigation the histology of the fœtal organs has not been worked out. Since syphilitic fœtuses are nearly always macerated, the autolytic changes in the organs ruin their histology; so, as regards syphilis, very little has been lost. The normal histology of the fœtal organs has yet to be worked out.

5. *Osteochondritis.*

The lower epiphysis of both femora and the sternal ends of some of the ribs were examined in all cases. These bones, after removal, were sliced through longitudinally with a stout knife and the epiphyseal lines were inspected for the characteristic thickening and irregularity. Simple naked-eye examination was relied on to settle whether or no syphilitic osteochondritis was present. Later on in the series, on the advice of Professor Turnbull, microscopical sections of the decalcified epiphyseal ends were examined also. Professor Turnbull has been kind enough to write, especially for this report, an account of the pathology of syphilitic osteochondritis. His account forms an original contribution of very great value to this difficult subject.

6. *The Weight and Histology of the Placenta.*

All placentas were weighed and measured and visible details noted, both surface and on section. From each placenta two

or three large pieces were taken for microscopical examination. The placentas were not examined for the spirochæta pallida. Former personal experience, as well as the writings of other workers, had amply taught that although spirochætes could sometimes be demonstrated in the placentas of syphilitic fœtuses, they were only present in very small numbers and were extremely difficult to find. A proper search means cutting many sections (Levaditi) from many blocks of tissue from the same placenta. Since, if spirochætes can be found in a placenta they will always abound and will be easily demonstrable in the fœtus, the immense labour involved in searching the placenta for spirochætes would have been out of all proportion to the results gained. But if it so happens that the placenta is the only pathological material available for investigation in a doubtful case of fœtal death (*i.e.*, in the case of a premature macerated fœtus, where toxæmia of pregnancy or retroplacental hæmatoma, or other well-known causes of fœtal death, can be excluded), it would be absolutely necessary to examine the placenta for spirochætes, if precision were aimed at; remembering always that failure to find them would not exclude syphilis. In such a case the investigator must prepare himself for a long piece of work. It is a good plan to divide the placenta into four approximately equal segments by means of two imaginary diameters, and to take six blocks of tissue from each segment; the blocks are distributed so that three are near the periphery, two half-way between the periphery and the middle of the placenta, and one near the middle. By adopting, or even extending, this plan, the placenta will be examined evenly. The blocks are prepared by Levaditi's method and several sections cut from each. The investigator may be rewarded for his pains after hundreds of sections have been studied.

The histological examination of the placenta often yields results of immense value; in about 65 per cent. of cases of fœtal syphilis, the placenta shows changes which, to the practised eye, are almost pathognomonic.

A full discussion of all these changes will be found in the section of this report which deals with the placenta, where they are dealt with under the three headings: (1) Naked-Eye Appearances; (2) Weight-Ratio of the Placenta; and (3) Histology of the Syphilitic Placenta.

IV. (2) The Diagnosis of Fœtal Syphilis.

The demonstration in the fœtal tissues of the spirochæta pallida is, of course, absolute evidence of syphilis. There is a great deal of other evidence, some of great and some of small value. This other evidence forms what may be termed the secondary attributes of fœtal syphilis. It may be stated at once that one of these secondary attributes (osteochondritis) may be regarded as pathognomonic; another, typical histological changes in the placenta, is highly reliable.

In an investigation such as this, which deals with a fairly large material, the opportunity arises of assessing the diagnostic value of the secondary attributes.

It is of the utmost importance to know the extent to which it is justifiable to make the diagnosis of fœtal syphilis in the absence of the spirochæte. Such a position may often arise; for example, the necessary skill or laboratory facilities may not be available for the purpose, the tissues may have become spoiled by decomposition, or the staining methods may have failed. Furthermore, though this is unusual, the spirochætes may be so thinly distributed that they are missed. Lastly, we cannot neglect the baffling, though not generally accepted, view that the spirochæte may exist in a granular or spore-like form, and may reside as such in the fœtal tissues.

An attempt will therefore be made to assess the value of these secondary attributes and to form a standard for the diagnosis of fœtal syphilis apart from the demonstration of the spirochæte.

From past experience, the chief secondary attributes of the syphilitic fœtus are well-known, and are as follows :—

(1) The maternal Wassermann reaction is positive.
(2) Syphilitic osteochondritis.
(3) Enlarged liver.
(4) Enlarged spleen.
(5) Increased weight of the placenta.
(6) Histological changes in the placenta.
(7) Histological changes in certain of the fœtal viscera.

To these must be added an *unequivocal* history of syphilis in the mother, and the birth of former *known* syphilitic fœtuses.

The extent to which these secondary attributes are distributed and the combinations in which they occur in fœtal syphilis, will be learned by studying the group of cases in which the spirochæte was found. There are 37 such cases. Next, the remaining 263 cases, in which no spirochætes were found, can be scrutinized, and those in which the same attributes occur can be studied. By this means an assessment can be made of the value of each, as well as of their combinations, as evidence of syphilis. It may then be possible to place certain of the spirochæte-negative fœtuses into one of three groups : (1) Certain syphilis, (2) Probable syphilis, (3) Possible syphilis.

A.—*The Spirochæte-Positive Fœtuses.*

In Table II. are displayed the details of these 37 fœtuses.

The following general points may be noted about this group.

External Appearance.

The syphilitic fœtus is usually born macerated; 35 in this group were macerated and only two were in a fresh state. The maceration may be of any degree, being solely dependent on the interval between death and birth; it is usually advanced, as

in syphilis there appears to be no particular stimulus to the expulsion of the fœtus after its death, as there is in certain other conditions. There was nothing in the external appearance of this group of fœtuses to distinguish them from non-syphilitic macerated fœtuses. When the syphilitic fœtus is born in a fresh state or in an early stage of maceration, as is most unusual, skin rashes may be distinguished.

Of the two fresh fœtuses, No. 176, a moderately premature fœtus, died from the effects of cranial injury received during labour; the mother had had a perineorrhaphy performed a year previously, the labour was precipitate, and the rapid birth of the fœtus caused an extensive tear of the resistant perineum. In this case the cause of fœtal death was not syphilis, except perhaps indirectly in so far as it may have been responsible for the premature labour and the birth of a fœtus with a soft easily-injured head. The other fresh fœtus, No. 246, was born in a premature and feeble state, and was covered with a petechial rash; the heart continued to beat feebly for a few moments,but no attempt was made at respiration. Only two fœtuses had deformities; Nos. 227 and 328 both had double talipes equinus.

Prematurity.

All the fœtuses were premature but one (No. 84). As regards intrauterine age calculated from the menstrual period, of the 28 cases in which data were available, 16 were of the age of 32 weeks or less; the youngest had not yet reached viability and was aged 26 weeks (No. 227). As regards length, the shortest was 33 cms. (No. 227); eight were from 35 to 37 cms.; twelve from 38 to 40 cms., and the remaining eight varied between 46 and 50 cms. It may be noted here that No. 227 is the one exception to the rule that the present investigation deals only with viable fœtuses; it is the youngest fœtus in which I have as yet found the spirochæte.

The Fœtal Viscera.

There was nothing of note in the external appearance or on the cut surface of the abdominal or thoracic viscera of the syphilitic fœtuses of this series to distinguish them from the non-syphilitic. A striking appearance on inspecting the abdominal contents *in situ*, is very often the great size of the spleen; but this is not constant, nor is it peculiar to syphilis.

Associated Complications of Pregnancy.

In five cases there were associated complications; one case of hydramnios (No. 84), one of albuminuria of pregnancy (No. 102), two of placenta prævia (Nos. 141 and 149), and one of retroplacental (accidental) hæmorrhage (No. 416). These must be regarded as coincidences. In both cases of placenta prævia the fœtus was macerated, so the placental separation cannot be regarded as the cause of death; the same probably holds good for the case of retroplacental hæmorrhage. In the case of

albuminuria it is, of course, impossible to say whether death was due to syphilis or to pregnancy toxæmia.

Signs of Syphilis, or History of Infection, in the Mother.

Only three of the mothers had clinical signs of syphilis. In No. 4 a rash and ulceration of the fauces were first noticed about three months before labour: in No. 31 there were condylomata of the vulva: in No. 84 several indurated small ulcers on the vulva were first noted three weeks before labour. The remaining 34 mothers showed none of the common clinical signs of syphilis. (It must be admitted that the clinical examination of these mothers was not complete, being confined to the surface of the body, the throat and the external genitals; the more occult clinical signs of old syphilis were not sought for, *e.g.*, examination of the fundus oculi and of the central nervous system.)

From only three other mothers was a clinical history of syphilis obtained. At the best, a history is not very reliable in a class of patient which is not in the habit of noticing or seeking medical advice for slight symptoms. One gave a clear account of an infection six years previously: the other two told of a discharge, soreness of the genitals and a sore throat coming early in the recent pregnancy. The absence of clinical signs or history in these cases is only one more example of what has often been emphasized before. Syphilis in women, in pregnant women especially, may be a very mild disease. The absence of signs or history is of no clinical value. It commonly happens that the birth of a syphilitic fœtus is the only evident symptom, and the first one to attract attention to the disease in the mother.

What I have already termed the secondary attributes of fœtal syphilis will now be considered. As stated, these are (1) a positive Wassermann reaction in the mother, (2) osteochondritis, (3) enlargement of the liver and spleen, (4) increased weight of the placenta, (5) histological changes in the placenta, (6) a history of syphilis in the mother, and of the birth of former syphilitic fœtuses. This will be dealt with in the section on the diagnosis of syphilis in spirochæte-free fœtuses (p. 35).

The Wassermann reaction was positive in 35 of the 37 cases (95 per cent.). The negative reactions found in the mothers of spirochæte-positive fœtuses came as a surprise; in one the reaction was repeated a week later and was again found negative. Such an occurrence is now well recognised by many observers, though it is admittedly rare and is hard to explain.

Osteochondritis.

The epiphyseal line (*i.e.*, the yellowish-red zone between the epiphyseal cartilage and the diaphysis) was found to be irregular in thirty-six of the 37 cases (97 per cent.). The irregularity and jaggedness of this line was accepted as the criterion of syphilitic osteochondritis. Routine microscopical examination was not undertaken. In view of what I learned from Professor Hubert

Turnbull, towards the close of my investigation, I regret this was not done, for irregularity of the line is an advanced stage of the process; at an early stage there is merely thickening of the line (or "zone of provisional calcification"). "In early lesions there may be no other recognisable abnormality: the junction of the epiphysis with the diaphysis, then, remains regular and unbroken." (*See* Professor Turnbull's account, on p. 41.) The single spirochæte-positive fœtus here noted as not having irregularity may have had the thickening of the early stage, and amongst the spirochæte-negative fœtuses certain early cases of osteochondritis may have been missed.

Enlargement of the Liver.

The section of this report, and the tables, dealing with the weight-ratios of the fœtal organs must here be consulted. For the macerated spirochæte-negative fœtuses the mean weight-ratio of the liver will be seen to be 35, and for fresh fœtuses 21. In contrast to this, the mean weight-ratio for the 35 macerated spirochæte-positive fœtuses is seen to be 22, and for the two fresh fœtuses 16. Taking each case individually of macerated and fresh spirochæte-positive fœtus, it will be seen that all the liver weight-ratios are below the mean for non-syphilitic cases: in fact, only one macerated spirochæte-positive fœtus has a ratio of over 30 (No. 148) and this fœtus was extremely macerated and the liver very deliquescent.

The livers of the 37 spirochæte-positive fœtuses are thus without exception above the average weight. But is this peculiar to syphilis? When we examine the ratios for the non-syphilitic fœtuses we find it is not. For amongst the 95 spirochæte-negative macerated fœtuses, it will be noted that there are 51 (including Nos. 12, 65 and 124, classed as "certain syphilis"), with a ratio of less than 35, and 43 with a ratio of 30 or less. Amongst the 162 fresh spirochæte-negative fœtuses we find there are 63 with a ratio of less than 20 (including No. 229, classed as "certain syphilis)." All the same, as will be further noted in the special section dealing with the matter, out of the 14 macerated spirochæte-free fœtuses which had *exceptionally* heavy livers, four were associated with a positive maternal Wassermann reaction, and one (No. 313) has been classified as "certain syphilis." The point that can be made from the foregoing is that enlargement of the liver is worthless as a diagnostic sign of syphilis, if taken by itself, for it is so often found in definitely non-syphilitic fœtuses; but when found in association with other secondary attributes of syphilis (such as positive Wassermann reaction and enlargement of the spleen) it may be helpful as an additional item of evidence.

Enlargement of the Spleen.

The special section and tables on organ-weights must again be consulted. The mean weight-ratio for the spleen amongst the 37 spirochæte-positive fœtuses is 188 (191 for the 35

macerated, and 134 for the two fresh). Compare with these the ratios for the spirochæte-free fœtuses; for the fresh fœtuses the ratio is 370, and for the macerated it is 485. It is evident that fœtal syphilis is characterized by enlargement of the spleen. Considering now the individual spleens of the spirochæte-positive fœtuses, it will be noted that all but one (No. 149) have a spleen above the normal mean weight; that is to say, an enlarged spleen was found in 97 per cent. (36 out of 37) of the spirochæte-positive fœtuses.

But is enlargement of the spleen peculiar to syphilis? A scrutiny of the ratios of the spirochæte-free fœtuses shows it is not; for 83 fresh fœtuses (including No. 229, classed as "certain syphilis") show a ratio of less than 350, and 42 macerated a ratio of less than 450. Furthermore, a number of these have *exceptionally* heavy spleens; for 11 fresh fœtuses show a ratio of under 200, and 18 macerated a ratio of under 300. But the interesting point emerges that amongst the above 11 fresh fœtuses there are two with a positive Wassermann reaction (Nos. 204 and 229) and amongst the 18 macerated there are six with a positive reaction (Nos. 65, 68, 211, 234, 290 and 313).

From the foregoing the value of enlargement of the spleen as evidence of fœtal syphilis may be assessed. It is practically constant, but is by no means peculiar. Taken by itself it is of small value, but when found in association with other secondary attributes of fœtal syphilis, it is helpful as an additional point of evidence. Enlargement of the spleen is more characteristic when accompanied by enlargement of the liver.

The Placenta.

The syphilitic placenta will be found fully discussed in the section of this report which deals with the placenta; only a brief summary will be given here of the conclusions reached as regards the value of increased weight and histological changes as evidence of syphilis.

1. *Increased Weight.*—Heaviness of the placenta is not a sign of great worth in the diagnosis of fœtal syphilis, because (*a*) amongst the non-syphilitic placentas, a fair number are as heavy as, or heavier than, the placentas of the spriochæte-positive fœtuses; (*b*) amongst the placentas of the spirochæte-positive fœtuses, a fair number are no heavier, or less heavy, than the placentas of the non-syphilitic fœtuses.

2. *Histological Changes.*—In the syphilitic placenta occur histological changes which are almost, but, so far as the present experience goes, not quite pathognomonic of syphilis. Of the 37 spirochæte-positive fœtuses, 26 placentas were examined microscopically; 16 (61 per cent.) showed typical changes, 6 were suspicious and 4 were negative. Of the spirochæte-negative fœtuses 91 placentas were examined; 5 showed typical changes, 10 were suspicious and 76 were negative. The

five placentas showing typical changes belonged to macerated fœtuses of which three were proved to be certainly syphilitic. The two remaining cases with typical placental changes showed no other evidence whatever of syphilis.

These findings can be thus summarized. In the microscopical examination of 117 placentas, 21 gave appearances typical for syphilis; of these 21, 19 were undoubted cases of syphilis, whilst in two there was no evidence of syphilis in the fœtus or mother. From this it can be stated that the diagnosis of fœtal syphilis from typical placental changes will be correct in 90 per cent. of cases. Thus, a typical section is highly reliable. When the histological changes are merely suspicious they are unreliable as evidence.

B. *Cases of Syphilis other than the* 37 *Spirochæte-Positive Fœtuses.*

Having noted the distribution of the secondary attributes of fœtal syphilis amongst the spirochæte-positive fœtuses, and having appraised to some extent their value, we are now in a position to apply that knowledge to the 263 spirochæte-negative fœtuses, in order to find out if there be any syphilitic fœtuses amongst them. The diagnosis of syphilis will have to be justified by noting secondary attributes of sufficient importance, or in sufficient combination, in the same case.

It has already been pointed out that mere enlargement of the liver and spleen is in itself of very little value, but is useful as an additional item of evidence if associated with other more important points. The three attributes of outstanding value are a positive maternal Wassermann reaction, osteochondritis, and typical placental changes; the importance of the first is self-evident; that of the second is generally recognised, and it has been pointed out that typical placental changes are likely to be correct in 90 per cent. of cases.

As regards the former history of maternal syphilis, or of former syphilitic dead-births, I give now the details of three cases. No. 313 is a most remarkable and important case, and No. 292 is hardly less important.

(1) Nos. 41 and 313 (two successive fœtuses with the same parents).—Fœtus No. 41 was undoubtedly syphilitic and will be found in the table of spirochæte-positive fœtuses. This fœtus was born on May 2nd, 1914. The mother again became pregnant and a dead macerated fœtus (No. 313) was born on May 25th, 1915. The maternal Wassermann reaction was again positive, but *there were no post mortem signs of syphilis in fœtus or placenta*, except enlargement of the liver and spleen. No spirochætes were found in the fœtal tissue, either in Levaditi sections or by the direct method; a most intensive search was made by both methods. This fœtus (No. 313) has been classed as "certain syphilis" because of the history of the case, though the signs of fœtal syphilis consisted only in enlargement of the liver and spleen.

(2) No. 292.—Premature macerated fœtus born on April 19th, 1915. The maternal Wassermann reaction taken on April 22nd, was negative. *There were no signs of syphilis in fœtus or placenta.* A most persistent search was made for spirochætes both by Levaditi and direct methods.

Very fortunately, I had the former history of this patient. She was under the care of my colleague, Dr. Arthur Whitfield, at King's College Hospital. She first came under his care in April 1914, for late secondary syphilis, and he gave her an efficient course of treatment until August 1914. Her Wassermann reaction was positive on April 30th, 1914, and in July 1914; in March 1915, it was negative and, as already mentioned, it was again found negative a few days after labour.

Was the cause of fœtal death syphilis? If the mother's former history had not been known, the answer would certainly be in the negative. I feel I have been justified in classing it as "probable syphilis," in spite of the total absence of post-mortem findings.

(3) No. 278.—Moderately macerated fœtus born in April 1915. No signs of syphilis in mother, fœtus or placenta. The mother had had 13 former pregnancies resulting in the birth of seven premature dead fœtuses and six living children. Four of the dead births occurred in sequence between the years 1903 and 1907. She gave a history of infection with syphilis in 1903—just before her series of dead-births. She was under treatment at the London Lock Hospital in 1903. In the year 1912 she got a recurrence of the disease and was again under treatment at the Lock Hospital. The two pregnancies in 1913 and 1914, immediately preceding the present one, resulted also in premature dead births.

Was syphilis the cause of death in this fœtus? Without a knowledge of the former history the answer would certainly be in the negative. I have classed the case as "possible syphilis."

From the 263 spirochæte-negative fœtuses emerge 25 which have to be scrutinized as possible cases of syphilis. They are set out, divided into macerated and fresh, in Table III. They comprise all the additional cases with a positive maternal Wassermann reaction, the three cases of osteochondritis, the five cases showing typical syphilitic placental changes, and the three cases with a clear history of maternal syphilis.

From this group of 25 cases, five stand out prominently, and by reason of the combination of important attributes each possesses, may be classified as undoubted cases of syphilis:—

No. 12, W +, osteochondritis, typical placenta.

No. 65, W +, osteochondritis, typical placenta, enlarged spleen.

No. 124, W +, typical placenta.

No. 229, W +, osteochondritis, enlarged liver and spleen.

No. 313, W +, enlarged liver and spleen, clear history of a former spirochæte-positive fœtus.

The standard adopted here is that the combination of a positive maternal Wassermann reaction, with osteochondritis or typical placental changes, or still more with both together, may be accepted as sure evidence of fœtal syphilis. Osteochondritis by itself is generally accepted as sufficient evidence: the fact that all the examples of osteochondritis in this series occurred in association with other evidence of syphilis reaffirms the accepted view that it is pathognomonic.

There still remain 20 cases, 17 with a positive maternal Wassermann reaction, two (Nos. 210 and 335) with typical placental changes only, and one (No. 292) with a very clear history of recent maternal syphilis. Some of them are accompanied by enlargement of the liver and spleen, or of both, and in one the placenta is suspicious of syphilis. They must all be regarded as cases of possible syphilis. But can any of them be placed in the more exalted category of "probable" fœtal syphilis? Any standard must of course be a purely arbitrary one: but as I am here particularly concerned with assigning the cause of fœtal death, I shall attempt to fix one. Otherwise, since there was no other apparent cause of death for many of these fœtuses, they will have to be dismissed into the category of "cause of death unknown." I regard a positive maternal Wassermann reaction alone, in the absence of other signs in a *fully investigated case*, as being insufficient evidence of "probable" fœtal syphilis. It has already been pointed out that "suspicious" histological changes in the placenta form unreliable evidence, the probability of syphilis being only 38 per cent.

The only attributes we are now left with are typical placental changes, enlargement of the liver and spleen and a clear maternal history.

Enlarged liver and spleen, when occurring together in the same fœtus, are very much more characteristic of syphilis than when they occur singly. They are certainly much more characteristic than "suspicious" histological changes in the placenta.

There are three of these fœtuses in which *exceptional* enlargement of the liver and spleen occur in combination (Nos. 234, 290 and 406*); these I shall class as cases of "probable syphilis." I shall also place No. 292 in the same class, because of the undoubted evidence of maternal syphilis immediately preceding and during the early period of pregnancy. Cases with typical placental changes I have also considered worthy of being classed as "probable," Nos. 210 and 335.

The remaining 14 may be classed as cases of possible syphilis; of these, nine have already a sufficient cause of death without invoking syphilis (Nos. 22, 80, 185, 370, 46, 163, 165, 167 and 204).

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* The ratios in No. 406 do not quite reach the standard I have laid down for *exceptionally* heavy liver and spleen, but they approach it

SUMMARY OF CASES OF FŒTAL SYPHILIS.

	Cases.
I. Spirochæte-positive fœtuses	37
II. Spirochæte-negative fœtuses—	
Certain syphilis	5
Probable syphilis	6
Possible syphilis	14

The 42 cases of proven syphilis amongst the 300 cases investigated makes the incidence of syphilis 14 per cent. If the six cases of "probable" syphilis are added, the percentage is raised to 16.

TABLE II.

SPIROCHÆTE-POSITIVE FŒTUSES.

No.	Age.	Wt.	Length.	W.R.	Sp. pallid.	Epiph.	Wt. liver.	Wt. spleen.	Wt. plac.	Placental histology.
4	32	1,940	39	+	+	+	80	14	360	?
14	?	1,990	38	+	+	+	148	16	360	Typical of syphilis.
21	?	1,090	36	+	+	+	90	17	330	Typical of syphilis.
29	30	1,470	38	+	+	+	87	9	280	Suspicions of syphilis.
31	?	1,470	38	+	+	+	88	8	470	?
40	?	800	35	+	+	+	31	3·5	260	Typical of syphilis.
41	?	2,700	49	+	+	+	123	9·5	370	Negative.
53	30	625	36	+	+	+	33	5	160	Typical of syphilis.
61	36	1,000	42	–	+	+	70	14	320	Typical of syphilis.
62	37	2,500	45	+	+	+	140	10	327	Typical of syphilis.
79	34	1,750	40	+	–	+	75	14	250	Typical of syphilis.
84	40	3,300	50	+	+	+	350	60	?	?
102	29	1,050	39	+	+	+	42	3	170	Negative.
109	?	1,100	37	+	+	+	168	10	225	?
118	30	1,250	36	+	+	+	91	9	220	Typical of syphilis.
129	34	1,550	46	–	+	+	63	13	220	Suspicions of syphilis.
132	31	1,400	43	+	+	+	57	7	190	Typical of syphilis.
141	28	750	35	+	+	+	33	4	185	Negative.
148	36	1,040	39	+	+	+	31	3·5	152	Suspicions of syphilis.
149	30	1,300	37	+	+	+	44	2	200	?
176	34	2,650	46	+	+	+	160	12	340	Typical of syphilis.
181	38	2,670	46	+	+	+	87	7	350	Typical of syphilis.
186	?	1,200	47	+	+	–	95	8	?	?
193	32	2,250	40	+	+	+	86	18	410	Typical of syphilis.
222	30	1,800	39	+	+	+	68	10	?	?
227	26	590	33	+	+	+	21	3	210	Suspicions of syphilis.
243	32	1.365	40	–	+	+	51	13	?	Suspicions of syphilis.
246	34	2,170	42	+	+	+	122	45	370	Suspicions of syphilis.
328	28	1,125	46	+	+	+	116	8	335	Negative.
332	30	2,000	41	+	+	+	153	20	520	Typical of syphilis.
342	?	2510,	45	–	+	+	106	28	225	?
377	38	2.020	47	+	+	+	118	28	400	Typical of syphilis.
391	?	1,240	40	+	+	+	73	10	?	?
396	34	2,420	44	+	+	+	96	12	?	?
399	28	1,200	36	+	+	+	60	11	?	?
413	34	1,100	39	+	+	+	52	4	280	Typical of syphilis.
416	32	1,840	41	+	+	+	97	20	500	Typical of syphilis.

TABLE III.

Spirochæte negative fœtuses, with positive maternal Wassermann Reaction or other secondary attributes of fœtal syphilis.

A. *Macerated.*

No.	Wt.	Length	W.R.	Sp. pallid.	Epiph.	Wt. liver.	Wt. spleen.	Wt. Plac.	Placental histology.	
12	1,430	39	+	—	+	55	2·5	280	Typical of syphilis	Classed as "certain syphilis."
22	1,920	42	+	—	—	45	3·5	?	Negative	Placenta prævia: "possible syphilis."
65	1,950	40	+	—	+	75	7	650	Typical of syphilis	Classed as "certain syphilis."
68	1,820	47	+	—	—	37	7	560	Negative	Cause of death unknown: "possible syphilis."
80	1,190	42	+	—	—	31	?	?	?	Albuminuria of pregnancy: "possible syphilis."
124	2,550	47	+	—	—	85	5	260	Typical of syphilis	Classed as "certain syphilis."
164	1,040	36	+	—	—	171	3	?	?	Cause of death unknown: "possible syphilis."
185	1,690	43	+	—	—	61	2	155	Negative	Small placenta: "possible syphilis."
211	2,700	41	+	—	—	66	9·5	340	Negative	Cause of death unknown: "possible syphilis."
234	2,200	42	+	—	—	148	34	?	?	Classed as "probable syphilis."
290	2,585	54	+	—	—	147	12	510	Negative	Classed as "probable syphilis."
313	2,890	45	+	—	—	143	10	385	Negative	Classed as "certain syphilis." Same mother had a spirochæte-positive fœtus, one year before.
370	2,050	44	+	—	—	103	4·5	230	?	Albuminuria of pregnancy: "possible syphilis."

TABLE III.—*cont.*

No.	Wt.	Length	W.R.	Sp. pallid.	Epiph.	Wt. liver.	Wt. spleen.	Wt. Plac.	Placental histology.	
210	1,120	41	—	—	—	20	2·5	290	Typical of syphilis	Cause of death unknown : "probable syphilis."
278	2,950	47	—	—	—	86	8	250	Negative	Classed as "possible syphilis," because of former history of mother.
292	2,160	45	—	—	—	42	4	400	Negative	Classed as "probable syphilis," because of history of recent syphilis in mother.
335	2,050	43	—	—	—	43	4·5	470	Typical of syphilis	Accidental hæmorrhage : "probable syphilis."
						B.—*Fresh.*				
46	—	—	+	—	—	72	4·5	320	Negative	Retroplacental hæmorrhage : "possible syphilis."
134	3,650	58	+	—	—	200	14	?	?	Difficult breech labour : "possible syphilis."
163	2,250	42	+	—	—	95	2·5	340	Suspicions of syphilis	Torn tentorium, cerebral hæmorrhage : "possible syphilis."
165	1850	48	+	—	—	105	5	?	?	Placenta prævia, cerebral hæmorrhage : "possible syphilis."
167	1,940	44	+	—	—	94	8	275	Negative	Retroplacental hæmorrhage : "possible syphilis."
204	2,590	51	+	—	—	115	15	270	Negative	Retroplacental hæmorrhage : "possible syphilis."
229	1,010	36	+	—	+	76	29	330	?	Classed as "certain syphilis."
406	2,672	51	+	—	—	178	12	445	Negative	Torn tentorium, cerebral hæmorrhage : classed as "probable syphilis."

IV.—(2) Recognition of Congenital Syphilitic Inflammation of the Long Bones.

By Hubert M. Turnbull, D.M., Director of the Pathological Institute of the London Hospital.

Syphilis in the fœtus or in infants may give rise to inflammation in the diaphysis, at a distance from the epiphysis, or in the periosteum. Much more commonly, however, it causes inflammation in the diaphysis at its junction with the epiphyseal cartilage—that is to say, in the metaphysis—and in the epiphyseal cartilage itself. This combination of metaphysitis with epiphysitis was called by Wegner (1870) "osteochondritis syphilitica."

If the cut surface of a longitudinal section of a fresh, *normal long bone* of a fœtus or child is examined with the naked eye, from an epiphyseal cartilage towards the diaphysis, the following zones can be recognised:—(1) white, flat, hard cartilage, corresponding to the portion in which growth in length and breadth of the cartilage proceeds; (2) a zone of more slaty and translucent, very slightly bulged, softer cartilage, corresponding to the portion of the cartilage in which, as the first preparatory changes for ossification from the diaphysis, the cells have multiplied, have arranged themselves in columns, and have enlarged at the expense of the matrix; (3) an opaque yellow, often almost white line, corresponding to that portion of the cartilage in which, as the final preparatory change, the matrix has undergone "provisional" calcification, but into which the vascular loops from the medulla have not yet advanced; (4) the deep red medulla of the diaphysis.

Further, on close inspection a few delicate vessels may be recognised in the cartilage close to the diaphysis. Microscopic examination shows that the cartilage is permeated by vessels which lie in canals. The vessels have the following arrangement: vessels pass transversely inwards from the perichondrium, at regular intervals which become wider as the diaphysis is approached, to anastomose in the mid-line; other vessels pass vertically from these transverse arches, uniting the transverse arches with one another and ultimately with vessels in the diaphysis. The vessels increase in size towards the diaphysis; the largest vertical vessels lie in the centre of the cartilage.

The colouration mentioned above becomes to a large extent obscured by hæmolysis in the macerated fœtus. The yellow *line of provisional calcification*, however, remains distinct. It is rendered more conspicuous by immersion of the bone in formaldehyde for a few minutes. This line is of the greatest importance as a guide to the presence of pathological processes. It stretches unbroken from one side of the bone to the other. It varies a little in depth, that is in measurement from epiphysis to diaphysis, at different ages, in different bones and in different portions of some epiphyses, being deepest where growth is most

active. Thus, at the upper end of the femur it is deeper beneath the neck than beneath the great trochanter. At its deepest, however, it is so shallow as to make measurement extremely difficult; its depth is almost invariably less than half a millimetre. Its epiphyseal and diaphyseal limits are even and regular; if it broadens, for instance beneath the neck of the femur, it broadens evenly without any step in either of these limits. The line may be absolutely straight, or show a single curve or more than one curve. In a section through the centre of a rib, for instance, it is frequently found to be of "bow shape," two convexities projecting towards the diaphysis on either side of a notch towards the epiphysis; this notch corresponds to the position of the large vertical vessels which pass from the centre of the epiphyseal cartilage to the diaphysis. When curved or sinuous, however, the curves are even, regular and unbroken.

1. *The effect of syphilitic osteochondritis* upon the above normal zones varies according to the degree of the injury to the normal tissues, by the spirochætes and their toxins, the stage and intensity of the inflammatory reaction, and the site, in cartilage or metaphysis, of the maximum of injury and inflammatory reaction. The effect is, therefore, manifold. The following are the most important changes seen under the microscope, together with their effects upon the normal pattern as seen with the naked eye :—

(1) Invasion from the diaphysis of the provisionally calcified cartilage by vessels, osteoclasts and osteoblasts may be checked, although the cartilage continues to grow in length and there is no cessation in the normal changes therein which, including calcification, are preparatory to its transformation into bone. This results, obviously, in the yellow zone of provisional calcification becoming abnormally deep. It may be two or more millimetres deep. *In early lesions there may be no other recognisable abnormality ; the junction of the epiphysis with the diaphysis, then, remains regular and unbroken.* When the increase in depth of the zone of provisional calcification is not great and is the only abnormality present, diagnosis even with the microscope, apart from the discovery of spirochætes, is very difficult. Fortunately, in most cases microscopical investigation reveals some degree of the other changes mentioned below, and thus simplifies the diagnosis.

(2) Whilst ossification from the diaphysis is checked in this manner in most of the epiphyseal-diaphyseal junction, it may continue at the sides of one or more of the vertical vessels which unite the cartilage and the diaphysis. At such spots the deep zone of provisional calcification becomes shallow, and, consequently, the diaphyseal border of the zone is notched and irregular.

(3) Owing to curtailment of the normal vascular supply, portions of the epiphyseal cartilage may become degenerate

and not undergo provisional calcification and subsequent ossification.

The vertical vessels, however, which unite the cartilage with the diaphysis are relatively large vessels, and the blood supply from these is less likely to be cut off. Provisional calcification and subsequent ossification usually proceed, therefore, round some at any rate of these. In consequence, narrow processes, or broader tongues, of red marrow project from the diaphysis into the epiphyseal cartilage and alternate with tongues of cartilage which project into the diaphysis. The line of junction of the epiphysis with the diaphysis then appears dentate, and the zone of provisional calcification follows the irregular line of the interlocking teeth.

Further, as growth proceeds, the level at which provisional calcification should normally take place may reach transverse chondral vessels of which the blood supply has not been interfered with. Round these vessels, in the epiphysis beyond the degenerate portions of cartilage, calcification and subsequent ossification take place in due course. This leads to the occurrence, in the epiphyseal cartilage at a distance from the main epiphyseal limit of the diaphysis, of red streaks and dots which are each bordered by a narrow zone of yellow calcification. Transverse chondral vessels have become more conspicuous to the naked eye, and are, moreover, surrounded by a visible zone of calcification. At a later stage are seen deeper transverse, red bands, bordered towards the epiphysis by a zone of yellow calcification. Now, not only is the junction of the epiphysis with the diaphysis dentate, but transverse zones of provisional calcification are seen at different levels. The disturbance of the normal pattern is extreme.

The cartilage, lying upon the deepened zone of provisional calcification, may not be interrupted by vertical vessels surrounded by provisional calcification. Then, calcification preparatory for ossification commences in the cartilage about a transverse vascular arch, at the time at which it should normally appear at the level of this arch. When this takes place, two zones of provisional calcification separated by a zone of cartilage are seen.

(4) Ossification may then proceed towards the epiphysis from this transverse vascular arch. In this way a more remarkable appearance is produced; a yellow, usually irregular, line of provisional calcification is followed by a zone of red marrow, in which osseous trabeculæ can be felt with the point of the scalpel, and this red marrow is separated from the red diaphysis by a second—the original—yellow, abnormally deep, zone of provisional calcification.

(5) Fibrosis is seen with the microscope in all but the slightest and earliest lesions. The fibrosis may affect the marrow of the metaphysis, or of the canals in the cartilage, or of both. In

advanced, intense inflammations a dense granulation tissue is formed. In the metaphysis, formation of such granulation tissue is associated with erosion by osteoclasts of trabeculæ of calcified cartilage and bone. When the cartilaginous canals are affected they become greatly widened. The granulation tissue may undergo caseation and softening. The epiphysis may become separated from the diaphysis by a transverse zone of granulation tissue in which trabeculæ of calcified cartilage and bone have been eroded; or the epiphyseal cartilage may be divided by a zone of granulation tissue which has widened the canals of an anastomosing, transverse vascular arch. If such granulation tissue undergoes softening, the epiphysis is separated completely from the diaphysis, or the epiphyseal cartilage is divided completely at a short distance from the epiphyseal-diaphyseal junction. Even if the granulation tissue has not softened, partial or complete separation at such spots is liable to occur during life or in the manipulation required for the removal of the bone.

Fibrosis is of the greatest assistance in the microscopic and macroscopic recognition of syphilitic osteochondritis. When slight fibrosis is present in the metaphysis, the marrow in the metaphysis appears to the naked eye more compact and paler than in the remainder of the diaphysis. The affected metaphysis still feels gritty when tested with the point of the scalpel. It may be pink or grey; it is frequently yellowish owing to the presence of numerous yellow trabeculæ of calcified cartilage in a grey fibrous matrix. The contrast with the remainder of the diaphysis is accentuated when the bone has been placed for a few minutes in formaldehyde. More advanced fibrosis appears slaty grey, or yellowish, gelatinous and slightly bulged, and feels soft owing to the erosion of calcified cartilage and bone. When slight fibrosis is present in the epiphyseal cartilage, the vascular canals are more conspicuous than normally. When advanced fibrosis is present, pink, grey or yellowish strands interrupt the cartilage in the sites of the canals.

In a macerated fœtus normal bones are, in the process of removal and section, very liable to break through the epiphyseal-diaphyseal junction; such separation must not be confused with separation due to fibrous granulation tissue.

The above possible alterations in the normal pattern of the junction of the epiphysis with the diaphysis may be summarised as :—deepening of the zone of provisional calcification; irregularity of the line of junction between the epiphysis and diaphysis; multiple zones of provisional calcification; enlargement of the chondral. vascular canals; fibrosis in the marrow of the metaphysis or of the canals of the epiphyseal cartilage.

Syphilitic osteochondritis in the bones of infants must be differentiated from *rickets*. Rickets also causes irregularity of the line of junction between the epiphysis and diaphysis and enlargement of the chondral canals. The essential abnormality

in rickets, however, is absence of calcification. Provisional calcification of the epiphyseal cartilage ceases, and, moreover, all bone formed during the disease remains "osteoid," that is to say, does not become calcified after its deposit. In the metaphysis of the long bones a zone of closely packed, osteoid tissue is formed. This is frequently yellowish in colour, but when tested with the point of a needle or scalpel it is found to be tough, elastic and free from grit, whilst the yellow deepened zone of provisional calcification in syphilis is hard and gritty. Further, all the bone becomes soft, owing to the formation of osteoid tissue whilst the old, true bone is removed in the process of growth. The whole bone, therefore, is cut with the knife with abnormal ease. In later stages the bone is pliable. In syphilis there is no such softening except in areas of fibrosis accompanied by erosion of bone. Such areas are focal and the granulation tissue therein can be recognised with the naked eye; the remainder of the bone remains hard and brittle.

II.—*Syphilitic Diaphysitis.*—Focal areas of syphilitic inflammation occur with considerable frequency in the medulla of the diaphysis at a distance from the epiphysis. The fibrosis is associated first with cessation of the deposit of bone and later with erosion of the trabeculæ of bone and calcified cartilage present. In early stages the areas of fibrosis are seen by the naked eye as paler, pink areas in the red marrow of the diaphysis; in later stages the granulation tissue is slaty blue or yellowish, frequently gelatinous, and the affected area feels soft.

III. *Syphilitic Periostitis.*—This lesion is rare, but may accompany advanced osteochondritis. It results in a layer of bone and red marrow, or rarely granulation tissue, being deposited outside the original corticalis. This reduplication of the corticalis can be recognised easily by the eye. It must be remembered that in rickets a similar zone of marrow and osteoid tissue may be formed outside the original corticalis.

In conclusion, congenital syphilitic disease of bone is not a general systemic condition, but is due to the local presence of spirochætes. The older the child, the fewer are the portions of bone affected. In the fœtus the infection tends to be widespread, but it is not necessarily universal. Further, when several bones are affected, the lesions are greater and more conspicuous in some bones than in others. It is advisable, therefore, in each case to examine as many bones as possible. The femur, tibia, humerus and ribs are sites of election; the femur and ribs are easy of access, and should certainly be examined in all cases.

For microscopical examination, except when it is desired to stain spirochætes, the minimum of decalcification compatible with section should be employed: recognition of the distribution of the provisional calcification in the epiphysis is as important in microscopical examination as in the examination with the naked eye. Fœtal bones, fixed in 4 per cent. formaldehyde and

embedded in paraffin or celloidin, can be cut on the microtome without decalcification. It is important to remember that the calcium in bone is not stained by hæmatoxylin unless the bone has been fixed for a week or longer in formaldehyde solution or in Müller's solution.

If the spirochætes have not been destroyed a certain diagnosis can be made with great rapidity by examination with dark-ground illumination of scrapings from the metaphyses of suspected bones which have not been placed in any fiaxtive.

SECTION V.

On Cranial Stress in the Fœtus during Labour and on the effects of Excessive Stress on the Intracranial Contents; with an Analysis of 81 Cases of Torn Tentorium Cerebelli and Subdural Cerebral Hæmorrhage.

(1) Some points in cranial mechanics.

(2) The anatomy of the dura mater septa, especially as regards the presence of stress-bands and stress-lines.

(3) The morbid anatomy of tears of the septa, as found in 81 fœtuses.

(4) The mode of origin and source of subdural cerebral hæmorrhage.

(5) Clinical part; analysis of the 81 cases.

(6) Table IV. Tabular Summary of the 81 cases.

The frequent discovery of tears of the dura mater septa, associated with subdural cerebral hæmorrhage, in fœtuses dying during the course of labour was one of the most striking features of the investigation. Out of 167 fresh fœtuses the tentorium cerebelli was found torn in 81 (48 per cent.), associated with tearing of the falx cerebri in five cases and with subdural cerebral hæmorrhage in all but six. Most were the result of breech or forceps delivery, or of delivery through a contracted pelvis, but a few resulted from apparently normal labour. These cases, with their more important details, are arranged in Table IV.

I am convinced that a proper understanding of the events which lead to these injuries, and of the manner in which they arise, will enable many cases of them to be avoided and fœtal mortality to be correspondingly reduced. I propose, therefore, to discuss at some length the forces which act on the fœtal head and the injuries they produce in the intracranial contents. In other words, to consider the stresses to which the head is subjected and the strains from which it suffers.

V.—(1) On some Points in Cranial Mechanics.

(a) *A General Consideration of Cranial Stresses and Strains.*

During its passage through the birth-canal the fœtal head is subjected to the action of compressive forces, which are the resultant between the powers of labour and the resistance of the maternal passages. The head is, therefore, in a state of stress during labour, and this state may be conveniently termed "cranial stress."

Cranial stress is a compound compressive stress which may be roughly regarded as consisting of two elements, (1) a general compression of the whole head, and (2) a simple longitudinal compression by opposite forces acting at the ends of the long diameter of engagement of the head in the pelvis. It is this element of simple longitudinal stress which is of such special importance in its effects on the cranium and intracranial contents, and which is chiefly responsible for alteration in the shape of the head and stretching of the septa of the dura mater. As the head may engage in any diameter lying between extreme flexion and extreme extension, this longitudinal stress may occur in one of many directions, but as regards their effects on the head these directions may be resolved into two, (1) antero-posterior; along the diameters lying between the occiput and the forehead, as in vertex and breech presentations, and (2) vertical, along the diameters lying between the vault and base of the cranium, as in face and brow presentations.

It is obviously impossible, during the course of labour in any given case, to analyse the exact nature of cranial stress, to measure accurately its intensity, or to predict its effects. For practical purposes no more can be said than that the stresses and their effects vary chiefly with, (1) the absolute amount of the forces acting on the head at any given time, (2) the direction in which these forces act on different parts of the head, (3) the degree of plasticity, or ductility, of the head, and (4) whether the forces attain their maximum effect suddenly or gradually. The only means of estimating the intensity of cranial stress is by observing its effects after birth. Cranial stress may be normal or excessive; even in the easiest labours the head is, of course, subjected to a certain amount of stress. Normal stress leaves no observable effect, or merely a moderate alteration in shape; excessive stress results in an excessive degree of moulding, or, as I shall show later, in the infinitely more important effects of overstretching and tearing of the tentorium cerebelli and falx cerebri, and rupture of certain blood vessels. Tearing of the tentorium and falx is clear evidence of excessive cranial stress and forms the best and simplest means of estimating, post mortem, the degree of stress to which the head has been subjected. Excessive cranial stress is responsible for fœtal death to an extent hitherto little suspected.

On the Effects of Cranial Stress.—The effect of stress is strain. Without entering into a technical discussion on strain it may be stated that all strains can be analysed into, (*a*) slides or shearing strains, (*b*) stretches. A slide consists in the change of shape of a body without change of size, the familiar illustration of which is to change the shape of a book from a rectangular to an oblique shape by pressing against the long edge of one of its covers. In fœtal heads which have been subjected to stress, both these classes of strain may be found; the moulding of the fœtal head is an example of a "slide," and the tearing of the septa of the dura mater is an example of a "stretch."

The effects of a given stress on a body depend, of course, on the material of which it is composed and on its construction. If we now consider the fœtal head as a body exposed to stress, and try to analyse the possible strains, we are at once faced by the extreme complexity, not only of the materials which compose it, but of its construction. It consists of a non-rigid shell, of a peculiar shape, composed of loosely-joined plates of pliable bone jointed on to a rigid base and strengthened by the attachment of the dura mater and its system of septa; the contents of this shell are partly solid and partly fluid. Further, these contents do not lie in an absolutely closed space; for a certain amount of blood and cerebro-spinal fluid can be squeezed out of it, whilst the termination of the hind-brain can be crowded down through the foramen magnum. Even were it possible, it is not my intention to discuss the behaviour of all this complexity under stress. I shall confine myself to three specific points in the structure of the head. These are (1) its plasticity, depending on the degree of ossification of the cranial bones, (2) the attachment of the septa of the dura mater to the cranial bones, and (3) the connection with the septa of certain blood vessels. On these structural features depend the production of the two special strains which form the subject of this section of my report, viz., stretching and tearing of the tentorium cerebelli and falx cerebri and the rupture of certain cerebral blood vessels. The sequence of events may be summarized as follows.

The first effect of stress is alteration in the shape of the head, or moulding; this necessarily involves alteration in the position, absolute and relative, and curvature of the cranial bones. This movement of the bones is simultaneously transmitted to, *and is resisted by* (this is important to appreciate) the attached septa of the dura mater, which appear to form a mechanical system especially arranged to limit the movement of the bones forming the cranial vault, and to resist a dangerous degree of alteration in the shape of the head. The resisting septa become tense and stretched, and altered in position and direction. Over-stretching results in tearing, the most common site for tears being in the tentorium cerebelli at its junction with the falx cerebri. Finally these changes in the septa are transmitted to the vein of Galen, whose fixed point is at the apex of the tentorium cerebelli; this

vein becomes stretched, kinked and engorged, so that either itself or some of its tributaries are ruptured, resulting in subdural cerebral hæmorrhage of greater or lesser extent. (Refer to Fig. 1.)

These deductions are based on evidence gained by the study of (1) the mechanical construction of the fœtal head, especially the system on which the septa of the dura mater are arranged, the disposition of their strengthening bands, their connection with the cranial bones and the way they are influenced by alterations in the shape of the head, and (2) the injuries to the septa and blood vessels found in the case of fœtuses whose heads have been subjected to excessive stress during labour. The remainder of this section consists in the elaboration of these points.

(b) *On Alteration in the Shape of the Head, or Moulding.*

I have stated that moulding of the head is essentially an example of a slide or shearing strain, and is brought about principally by the action of compressive forces acting at the ends of the long diameter of engagement. (It is not strictly correct however, to state that the head does not change in size, there is probably a decrease in volume, brought about partly by the squeezing out of some blood and cerebro-spinal fluid and partly by the crowding down into the foramen magnum of the termination of the hind-brain. Evidence of such decrease in volume may be seen in the compensatory overlapping of the bones of the vault. I wish to emphasize that any decrease in the volume of the head by these means can be so slight as to be, for practical purposes, negligible. The alteration in shape is the all-important factor.)

Alteration in shape is brought about partly by a displacement of the bony vault as a whole and partly by bending of the individual bones which compose it. The displacement of the bones as a whole takes place chiefly at the junction of the plate with the base of the occipital bone; this hinge-joint allows a considerable range of movement backwards and forwards. The greatest bending of individual bones occurs in the parietals and frontal; the occipital bone is more rigid and, although it becomes bent to a certain extent, confines itself chiefly to the *rôle* of moving backwards or forwards on the occipital hinge, carrying with it the rest of the vault. The vault of the skull is plastic as a whole and can, within limits, change shape in most directions, but alterations in the shape of the head are chiefly dependent on the backward or forward movement of the occipital plate at the occipital hinge; bending of the bones is secondary to this and is, of course, a very necessary accompaniment.

According as to whether the forces acting on the head force the occipital bone forwards or backwards, alterations in the shape of the head may be divided into two groups, (1) decrease in antero-posterior measurement, (2) increase in antero-posterior measurement.

Decrease in antero-posterior measurement is brought about by pressure applied at the opposite ends of the diameters lying between the occiput and forehead, *i.e.*, suboccipito-bregmatic, suboccipito-frontal and occipito-frontal; such pressure occurs during labour in vertex presentations and in the after-coming head in breech presentations, and is correspondingly increased when the pelvis is contracted. Decrease in antero-posterior length is naturally accompanied by a compensatory increase in vertical height; in this form of moulding the chief change is a displacement forwards of the plate of the occipital bone, accompanied by arching of the vault of the head along the sagittal suture and free bending of the parietal and frontal bones. This is illustrated in Fig. 2 (ii).

Increase in antero-posterior measurement occurs when the head is compressed between the base of the skull and the vault, as in face and brow presentations. The chief change is a displacement backwards of the plate of the occipital bone; the vault of the skull becomes flattened, and the forehead is made protuberent by bending of the frontal and parietal bones. This is illustrated in Fig. 2 (iii).

(c) *On the Effect on the Dura Mater Septa of Alterations in Shape of the Fœtal Head.*

It is probable that, normally, the septa are in a state of rest, and are nowhere in a state of positive tension or slackness. When the head undergoes changes in shape, alterations in the tension of the septa are inevitable, owing to the nature of their attachments to the cranial bones. The behaviour of the septa is easily demonstrated by the following observations.

Take a fœtal head, reflect the scalp and remove sufficient of one parietal bone to allow the corresponding cerebral hemisphere to be removed and the septa to be viewed from that side. Now compress the head in the occipito-frontal diameter so that the head is shortened antero-posteriorly by the forward displacement of the occipital bone, and heightened vertically by the elevation and bending of the vault. (Refer to Fig. 1.) At the same time observe the septa. The middle two-thirds and the inferior free edge of the falx cerebri, and the tentorium cerebelli will be seen to become stretched and tense. At the same time the septa move as a whole, as can be appreciated by observing the anterior point of junction between the falx and tentorium; this will be seen to be displaced upwards and slightly forwards, the upward movement being much more pronounced than the forward. If, now, the compression be continued, the tension increases and finally the tentorium becomes overstretched and tears near its free border just below its junction with the falx. (Refer to Fig. 2.)

The manner in which this sequence of events occurs is this. That part of the cranial vault which becomes elevated and bent

gives attachment to, approximately, the middle two-thirds of the falx. This part of the falx is, therefore, pulled upwards by the vault; but it is attached below to the tentorium cerebelli and is thereby prevented from accompanying the vault upwards. In consequence, the middle two-thirds of the falx becomes tense and stretched. The pull from the falx is transmitted to the tentorium, which in its turn also becomes tense and stretched. This result is radically different from what might on first thoughts be expected to occur, for owing to the approximation of its posterior (occipital bone) to its anterior (crista galli) attachment, the falx might reasonably be conceived to become slack instead of tense. The true conception of what should occur only becomes clear when it is recollected that the part of the falx attached to the elevated vault is anchored down by the tentorium cerebelli.

Next compress the head between the vault and the base, as occurs in face and brow presentation, so that it becomes lengthened antero-posteriorly by the backward displacement of the occipital bone. The free border of the tentorium and the lower free border of the falx will be seen to become stretched and tense; the remainder of the falx becomes slack, owing to the approximation of its attached and free border by the lowered elevation of the vault of the cranium.

The septa should be regarded as stays to the cranial bones, like the stays of a mast. Excursion of the bones and bending are resisted by the tension of the septa, just as the swaying of a mast is resisted by the tension of its stays. If excursion and bending of the bones are too great the septa tear, just as the stays of a mast will snap if it is forced to sway too much. Carrying the simile a stage further, the breaking of a mast's stays allow it to oversway and break; the tearing of the septa permits still further displacement and bending of the cranial bones which brings about dangerous disturbances in the relationship of the intracranial contents.

According to this conception the septa exert a protective function in labour; they form a defensive system against excessive alterations in the shape of the fœtal head. Moulding of the head is, up to a point, a very beneficial process, without which many a fœtus could not be born; but excessive moulding is dangerous and may cause fœtal death by producing intracranial disturbances, the most evident of which is, as I shall later show, cerebral hæmorrhage from the overstretching and rupture of certain blood vessels.

Striking confirmation of the theory that the septa are designed to take stress is found in the fact that they contain special strengthening bands and fibres, arranged on admirable mechanical principles along the lines where stress is likely to fall during moulding of the head. I shall now describe these stress bands and stress lines in detail.

V.—(2) On the Anatomy of the Septa, especially as regards the Stress-bands and Stress-lines.

The Dura Mater consists of two layers, an inner and an outer, which differ widely in structure and function, and, no doubt, in origin. The outer layer is a tough fibrous membrane lining the inner surface of the cranial bones: it is in reality their inner periosteal layer and will subsequently be referred to as the periosteal layer. This layer is the important one from the point of view of the present subject: it sends into the cranial cavity the well known septa, and is channelled by the large venous sinuses.

The inner layer forms an inner lining to the periosteal layer and its septa; it is a thin transparent membrane resembling a serous membrane, formed of white fibrous tissue and elastic fibres and lined by a single layer of epithelium. Its function is to serve as a smooth, slippery membrane for the brain to rest on and to impinge against with the least possible friction.

The Periosteal Layer projects into the cranial cavity four sheets or septa of fibrous tissue, (1) the falx cerebri, (2) the two halves of the tentorium cerebelli, (3) the falx cerebelli. These septa take origin along the course of the great venous sinuses, which are themselves channels formed in the substance of the periosteal layer. The line of origin of the falx cerebri follows the course of the superior longitudinal sinus, that of the tentorium cerebelli the lateral sinus and that of the falx cerebelli the occipital sinus. The septa meet opposite the position of the greatest sinus of all—the torcular Herophili. Two points are here of importance; the membrane is especially strong where it forms the walls of the sinuses, and opposite the sinuses the membrane is firmly adherent to the bone, whereas elsewhere it is readily separable. Where the falx cerebri and tentorium cerebelli meet, they blend together and form a strong band or line. This line may, for descriptive purposes, be termed the *white line*. The *white line* is situated beneath the straight sinus and occupies the angle where the two halves of the tentorium meet. Its position may be gathered by referring to Fig. 4, where the black dotted line indicates the floor of the straight sinus, which runs backward in a channel formed in the falx cerebri. The white line pursues a straight course for the anterior two-thirds of its length, making an angle of about 45° with the horizontal; for the remainder of its course it runs more horizontally, following closely the floor of the straight sinus, and ends in a thick band or mass of fibrous tissue attached to the occipital bone in the region of the torcular Herophili.

These septa of the periosteal layer are not of equal strength and thickness throughout, but, at certain definite places, there is an aggregation of fibres into strengthening bands. *These bands are, I believe, developed to enable the cranium and its contents to withstand the effects of excessive stress during labour.*

They are certainly disposed on admirable mechanical principles in the particular parts of the septa which are most likely to have to bear the most strain. The tentorium and falx no doubt serve other functions than that of protecting the fœtal cranium and its contents from the effects of excessive stress, but I am convinced that this protective function is of the highest importance. Precisely how these bands are strained during the stress of labour and how they exert their protective functions I shall discuss later on, after I have finished the anatomical description.

According to the main directions in which they run, these strengthening bands may be roughly, but I believe usefully, divided into two main systems :—

(1) The antero-posterior system.
(2) The vertical system.

To illustrate their arrangement I have had two semi-diagrammatic drawings made. Fig. 3 is a fœtal head, bisected in the sagittal plane, the line of section passing immediately to the left of the falx cerebri; it illustrates the arrangement of the antero-posterior system. Fig. 4 is from a fœtal head, after removal of the parietal and part of the frontal and occipital bones, so as to expose the tentorium and falx in their entirety. In both these drawings the chief aggregation of fibrous tissue, in strengthening bands, appears white.

1. *The Antero-posterior System.*—In the region of the torcular Herophili is a mass of fibrous tissue, firmly attached to the occipital bone; it is broken up, and its arrangement complicated by being channelled by the torcular and the terminations of the superior longitudinal, straight and occipital sinuses. It seems to have three components: (1) the central and densest part is a wedge-shaped mass, firmly adherent to the occipital bone opposite the occipital protuberence, and lying below and to one side (generally to the left) of the torcular; (2) the upper part is a strong band which at first lies free, like a pillar, in the lumen of the superior longitudinal sinus; it passes downwards and forwards, in front of the torcular, to join the central mass; (3) its lower part is a broader but thinner collection of fibres disposed around the occipital sinus, forming the falx cerebelli. This main mass of fibrous tissue forms the meeting point of all four septa of the dura mater; it is the sheet-anchor of the whole complicated system.

The chief continuations forward of the main mass consist of two extremely important bands of considerable strength. These two bands are symmetrically placed on either side and originate fanwise from the sides of the main mass; their fibres converge as they pass forwards to be inserted into the anterior clinoid processes; they run along the lower surface of each half of the tentorium, and form the straight part of its free edge. They may be termed the *inferior horizontal bands of the tentorium*, and

play an important part in resisting excessive cranial stress. They are depicted in Figs. 3 and 5.

2. *The Vertical System.*—This system illustrates admirably the mechanical principle of stress-lines, and is represented in Fig. 4. It consists of two opposing sets of convergent fibres which meet in their points of convergence. From above downwards converge fibres from the falx cerebri; from below upwards converge fibres from the tentorium. From the falx the fibres originate from its middle two-thirds; from the tentorium the fibres originate chiefly from that part which covers the lateral sinus. The converging fibres meet in a strong broad band whose centre, roughly, is placed along the anterior fourth of the white line. This band may be called the *vertical band of the tentorium.* The main part of the band belongs to the tentorium and forms the curved part of its free edge; as it curves downwards and forwards it blends with the horizontal band of the tentorium. This band is subjected to strain in almost every labour. It is stretched when the head is elongated vertically.

I wish to direct particular attention to the triangular part of the tentorium which is bounded above by the white line, below by the line of the horizontal band of the tentorium and in front by the curved free edge of the tentorium. It includes the anterior vertical band of the tentorium. It is the part of the tentorium most commonly subjected to strain and most commonly torn.

The tentorium cerebelli, from the above description, is seen to be strengthened by two main sets of fibres, (*a*) a superficial set which sweeps upwards and inwards to be inserted into the white line and the anterior vertical band, and (*b*) a deep set which consists of the horizontal band. It frequently happens that when the tentorium is overstretched the tear consists in the upward separation of the anterior vertical from the horizontal band, instead of a simple tearing through of the anterior vertical band. This is illustrated in Figs. 10 and 11.

V.—(3) On the Morbid Anatomy of Tears of the Septa as found in 81 Fœtuses.

Passing now from theory to facts, do the facts, as ascertained by the circumstances in which the tears arise and the positions they occupy, in the septa of dead fœtuses, support the theory? I think they undoubtedly do. I have pointed out that as the head changes in shape, as in moulding, tension is thrown on the septa; that such tension occurs along definite lines of stress, strengthened by additional fibres and bands; that when the alteration in shape and consequent tension become excessive, the septa become overstretched and tear; and that the tears will naturally occur in that part of the septal system which is included within the area of stress and along the line of greatest stress.

When the tentorium cerebelli tears, the tear, as would be expected, nearly always is found in the triangular area I have described as situated beneath the line of junction of the tentorium and falx, and bounded in front by the anterior vertical band.

The tears may affect one or both sides of the tentorium, and may be complete or incomplete. Complete tears involve both the superficial and the deep layers of the tentorium; incomplete tears involve only the superficial layer, either the vertical band or the fibres posterior to it. Complete tears usually involve the free border of the tentorium; sometimes the free border is left intact, by reason of the extra strength it gains from the anterior vertical band, and the tear is in the nature of a perforation. In the latter case, the vertical band is always found hanging slack, which shows that it has been stretched beyond the limit of elasticity, and often contains a small hæmatoma. A not uncommon variety of tear is when the superficial layer, including the vertical band, is separated from the deep layer, including the horizontal band; this form of tear necessarily involves a certain amount of tearing through the free border of the tentorium. Very commonly a hæmatoma is found in that region of the tentorium which usually tears; this, I believe, is a sign of excessive stretching without tearing.

All these varieties of tear are shown in the illustrations. Fig. 6 (Case No. 339) shows bilateral complete tears, that on the left side being a perforation. Fig. 7 (Case No. 87) shows a complete tear on the right side involving the straight sinus. Fig. 8 (Case No. 340) shows a large perforating tear on the right side. Fig. 9 (Case No. 220) shows extensive bilateral tears, with rupture of the wall of the posterior end of the left lateral sinus. Figs. 10 and 11 (Case No. 265) show the inner and outer aspects of a tear involving separation of the anterior vertical from the deep horizontal bands; the vein of Galen was torn in this case. Fig. 1 (Case No. 420) shows an incomplete tear through the base of the anterior vertical band, and Fig. 12 (Case No. 179) shows an incomplete tear of the superficial fibres below and behind the anterior vertical band.

A summary of the tears is as follows :—

I. Bilateral, 64 :

(*a*) both complete - - - - -	35
(*b*) both incomplete - - - - -	15
(*c*) one complete and one incomplete -	14

II. Unilateral, 17 :

(*a*) complete - - - - - -	8
(*b*) incomplete - - - - - -	9

Tearing of the falx cerebri was found, always in association with tearing of the tentorium, in only five cases (Nos. 87, 179, 220, 305, 356). The tears, one or more in number, lay in the middle two-thirds of the falx, and were elliptical in shape, with

the long axis of the ellipse lying, as would be expected, in the line of stress.

V.—(4) On the Mode of Origin and Source of Subdural Cerebral Hæmorrhage.

Subdural cerebral hæmorrhage, varying in amount from a few drops to a copious hæmorrhage, was found in all but six cases (Nos. 179, 263, 302, 317, 338, 404). The hæmorrhage may be classed as severe, moderate or slight. Severe hæmorrhage accompanies a higher proportion of bilateral complete than of other varieties of tear, but there is no constant relation between the extent of the tear and the amount of hæmorrhage, as the following table shows :—

——	Severe.	Moderate.	Slight.	Absent.
Bilateral, both complete, 35 cases	21	11	1	2
Bilateral, mixed, 14 cases	7	5	1	1
Bilateral, both incomplete, 15 cases	4	7	2	2
Unilateral, complete, 8 cases	2	4	2	0
Unilateral, incomplete, 9 cases	2	3	3	1

The hæmorrhage does not originate from the tear itself. The fact that in six cases there was no hæmorrhage whatever, and that, in a few other cases, the tear itself, occupied by a tiny clot, lay entirely outside the boundaries of a fair-sized subdural hæmorrhage, is good proof of this. The tentorium cerebelli contains only a few fine capillary vessels (excepting, of course, the lateral sinus).

It was seldom possible, except in the few cases where a large sinus or blood vessel had ruptured, to find the exact source of the hæmorrhage; the lesser cerebral vessels are so small and delicate, and the fine arachnoid connective tissue surrounding them becomes so infiltrated with blood, that it is impossible to isolate them with accuracy from the surrounding hæmorrhage.

In case No. 220 the lateral sinus, in case No. 87 the straight sinus and in case No. 265 the vein of Galen were found ruptured; in these cases the hæmorrhage was enormous, and its source obvious. Failing the detection of the ruptured vessel, a valuable clue to the origin of the hæmorrhage is its exact situation in the subdural space and its relation to surrounding structures, especially blood vessels. In order to determine this more accurately I made small openings in some of the heads in which I expected to find hæmorrhage and soaked them in formalin solution for a month before dissection. A large hæmorrhage is not helpful for this purpose as its spread is too

diffuse, but a small hæmorrhage is necessarily confined to a small area of the subdural space. It soon became apparent that the origin of the hæmorrhage could be pinned down within narrow limits: for it was found that, in cases of cerebral hæmorrhage, a certain situation in the subdural space was never free from blood, and that, in the case of small hæmorrhages, the blood was always confined to this one situation and was in relation to the same blood vessels. This situation is a narrow area bounded in front by the transverse fissure, behind by the upper part of the tentorium, below by the pons Varolii and laterally by the mesial aspects of the anterior lobes of the cerebellum; its centre corresponds to the course of the vein of Galen. The blood vessels which occupy this area are the vein of Galen and its tributaries. As these vessels are precisely the ones that are likely to be stretched and torn, and as in two cases I found the vein of Galen itself ruptured, it is reasonable to draw the general conclusion that these vessels are the origin of the hæmorrhage in cases of excessive cranial stress.

The mechanism which leads to stretching and tearing of these veins must now be considered. Any movement of the apex of the tentorium is necessarily transmitted to the vein of Galen, whose fixed point is at its entrance into the straight sinus. In the common forms of cranial stress, *i.e.*, antero-posterior longitudinal stress, it has already been pointed out that the apex of the tentorium is drawn upwards, and that this upward displacement is very considerable in cases of excessive stress. The vein of Galen follows this movement with two results: (1) its line of direction is altered, and (2) it becomes stretched and tense. The altered course of the vein is admirably shown in Fig. 1; instead of passing backwards and slightly upwards, the vein is seen to be running almost directly upwards and making an acute angle with the straight sinus. In consequence, the vein becomes kinked at its entrance into the sinus, with the result that the flow of blood is impeded or obstructed. The enormous distension and engorgement of the vein of Galen and its tributaries is always a striking appearance in cases of torn tentorium and, together with the anatomical findings, is sufficient evidence of the occurrence of the series of events I have described. If, now, the distended veins are still further stretched, they rupture; it is sufficiently obvious that a vein, whose walls are already in a state of high tension from engorgement, will more easily rupture, as the result of further longitudinal stretching, than a vein whose walls are in a normal state of tension.

It now remains to consider which particular veins are ruptured. The vein of Galen itself is seldom ruptured: the rupture of such a prominent vessel could seldom escape careful observation, and I was only able to detect it twice. In these cases the vein had yielded at its fixed point, *i.e.*, at its point of entrance into the straight sinus. The tributaries which the

vein of Galen receives, from the point of its formation by the junction of the two internal cerebral veins to its termination in the straight sinus are (*a*) the two basal veins, (*b*) veins from the superior surface of the cerebellum, which run forwards and upwards to join the vein of Galen, (*c*) veins from the mid-brain, (*d*) veins from the pons. Another set of veins, although they do not enter the vein of Galen, must also be considered: these are veins passing from the superior surface of the cerebellum directly into the straight sinus. With the exception of the basal veins, the above veins are too small and delicate to be satisfactorily traced in the fœtus, unless they are distended with blood. I believe, so far as my observations go, that the usual source of hæmorrhage in these cases is from the tributaries which the vein of Galen receives from the cerebellum and mid-brain, and from the cerebellar veins which enter the straight sinus.

The mechanism of stretching and rupture of these veins is as follows. They are stretched between their two fixed points; their upper fixed point is the vein of Galen, and their lower is the cerebellum. The vein of Galen, as already described, is displaced upwards and forwards, and is kinked, by the pull of the tentorium. The cerebellum is not only held down, but is pressed still further down into the posterior fossa, by the downward pressure of the posterior poles of the cerebral hemispheres. In consequence of the upward pull and kinking of the vein of Galen, and the fixation or downward pull of the cerebellum, these delicate veins become stretched and engorged, and finally ruptured. The same mechanism of rupture applies to the cerebellar veins which enter the straight sinus direct.

V.—(5) Clinical Part: Analysis of the 81 Cases.

In macerated fœtuses the dura mater is so softened and weakened and the loosened cranial bones allow of such extreme alteration in the shape of the head that tears of the tentorium and falx are seldom absent, and arise on very slight provocation.

Out of the 167 fresh fœtuses, the tentorium cerebelli was torn in no less than 81, or 48 per cent. This demonstrates how extremely common the injury is. Tearing of the falx cerebri is comparatively rare, and was found, always in association with tearing of the tentorium, in only five cases (Nos. 87, 179, 220, 305 and 356). Subdural cerebral hæmorrhage, varying in amount from a few drops to a copious hæmorrhage, was associated with the tears in all but six cases (Nos. 179, 263, 302, 317, 338 and 404).

For purposes of clinical analysis the 81 cases found in the 167 fresh fœtuses may be divided into two main groups—

I. Those in which the fœtus was delivered by the head, 46 cases.

II. Those in which the fœtus was delivered by the breech, 35 cases.

I. The 46 head first deliveries comprised 44 cases of vertex presentation; one case of face presentation in which labour ended naturally (No. 414), and one case of brow presentation, in which labour was terminated by forceps (No. 220).

Analysing further the 44 cases of vertex presentation it will be seen that 25 were cases of forceps delivery and that the other 19 were cases of natural delivery.

(1) *Forceps Cases.*—The indications for the use of forceps were as follows :—

(*a*) Contracted pelvis, seven cases. The low operation was used in four cases (No. 175 after pubiotomy) and the high in three (No. 231 was followed by Cæsarean section).

(*b*) Placenta prævia, four cases. The low operation was used three times and the high once.

(*c*) Prolapsed cord, four cases. The low operation was used three times and the high once.

(*d*) Prolonged second stage of labour. In six the delay was due to persistent occipito-posterior presentation; in one of these the fœtus was extremely large (No. 422). In four it was said to be due to uterine inertia.

In considering these forceps cases it is apparent that in some the force exerted was unavoidably great; such are all the cases of high forceps operation (always accompanied by a high fœtal death-rate), of pelvic contraction, of prolapsed cord, and those cases of placenta prævia in which the fœtus was delivered rapidly. In such cases tearing of the tentorium is readily accounted for.

On the other hand, in cases where forceps is applied merely for a prolonged second stage due to malposition of the head or inertia of the uterus, the occurrence of the tearing of the tentorium invites criticism. The production of sufficient cranial stress to make the tentorium cerebelli tear means either that the force exerted by the forceps was excessive or was applied to the wrong diameter of the head, or both; neither should occur if the os is fully dilated or if the occiput is directed forwards. If the forceps is responsible for the saving of many fœtuses, it is also responsible for the unnecessary death or injury of many others; it is used too often and on the flimsiest indications.

The mechanical action of the forceps in producing a dangerous degree of cranial stress involves two separate considerations.

The Amount of Force exerted.—If the force exerted be sufficiently great, especially in the case of premature heads, the degree of stress produced will be enough to make the tentorium tear, even if the forceps be applied in the best possible manner.

The Direction of the Applied Force.—The most dangerous direction is when the forceps is applied to the antero-posterior diameter of the head, or to one approximating it. Even a moderate amount of force applied in this direction can, by causing vertical elongation of the head, lead to stretching and

tearing of the tentorium. The risk of applying forceps to the extremely plastic head of a premature fœtus is well known.

(2) *Natural Delivery in Vertex Presentation.*—In some of these cases the conditions were favourable for the occurrence of excessive cranial stress; in others it is puzzling to find the reason. The 19 cases may be analysed as follows :—

Normal vertex presentations, three cases (Nos. 364, 400, 421).
Twins (one fœtus lived in each case), three cases (Nos. 156, 196, 341).
Placenta prævia, three cases (Nos. 338, 353, 360).
Accidental hæmorrhage, four cases (Nos. 191, 293, 317, 390).
Long second stage (occ. ant.), one case (No. 312).
Rapid labour following induction for albuminuria of pregnancy, one case (No. 398).
Precipitate labour, one case (No. 369).
After colpoperineorrhaphy, one case (No. 176).
General œdema of fœtus, one case (No. 212).
Persistent occipito-posterior, one case (No. 406).

Two were cases of normal full time labour (Nos. 400 and 421), *i.e.*, the second stage was not too rapid or too long, the resistances were not unduly great, the occiput was forwards and the fœtus was neither premature nor of great size. In others, prematurity of the fœtus, with the consequent greater plasticity of the cranium, is the only predisposing factor to be found; such are one of the cases of normal labour (No. 364), the cases of twins—in No. 156 the head was born with the occiput behind—two of the cases of placenta prævia (Nos. 338 and 360), and three of the cases of accidental hæmorrhage (Nos. 293, 317 and 390). A very rapid second stage, unless the resistances be slight, and especially if the fœtus be premature, is an easily appreciated factor; such are cases 176, 369, and 398. It is to be noted that pituitrin was given to one of the cases of placenta prævia (338, fœtus premature), and to one of accidental hæmorrhage (191); the action of this powerful ecbolic as a possible factor in producing excessive cranial stress should not be overlooked. Case 176 is an admirable example of a combination of many factors: the mother had an extensive colpoperineorrhaphy done a year previously for prolapse of the uterus; the premature fœtus (syphilitic) was expelled with great rapidity, causing severe laceration of the thick resistant perineum.

It is not at all easy to give a reasonable explanation for the torn tentorium in many of these cases of natural vertex delivery. I strongly suspect that over-zealous guarding of the perineum from rupture, especially by strong forward pressure against the perineum forcing the occiput against the symphysis, may explain some. Rupture of the perineum is held by many to

be a disgraceful occurrence and a disaster above all others to be avoided.

II. *Breech Deliveries.*—Tears of the tentorium cerebelli are found with great frequency in dead fœtuses delivered by the breech. There are 47 cases of breech delivery amongst the 168 fresh fœtuses, and I have found tearing of the tentorium in 35 or, approximately, 75 per cent.

The explanation is simple, and I have already referred to it. During its final passage the after-coming head is rapidly, though compratively momentarily, compressed in a series of diameters lying between the forehead and the occiput; compression in this direction causes vertical elongation of the head (*see* Plate 20), and I have already pointed out that it is this particular alteration in shape which throws the greatest strain on the tentorium cerebelli.

Analysis of Cases.—Cases of breech labour present a wide divergence in type. In order to appreciate the significance of tears of the tentorium in breech delivery it is necessary to classify the cases. Where the after-coming head is dragged through a contracted pelvis, or where the fœtus has had to be delivered rapidly for such complications as antepartum hæmorrhage, the discovery of the effects of excessive cranial stress might reasonably be anticipated. But tearing of the tentorium is by no means confined to cases of this type. It is a significant fact, and one of great practical importance, that many are cases of what may be called ordinary normal breech labour.

Breech labour may be divided into three classes :—

(1) Primary uncomplicated breech presentation, in which there are no complications of pregnancy, and no complications of labour (except premature labour) other than those arising direct from the presentation, such as extended legs or arms, prolapse of the cord.

(2) Primary complicated breech presentation, such as breech presentation in cases of antepartum hæmorrhage, contracted pelvis, etc.

(3) Breech by version.

The 47 cases of breech delivery, and the 35 with tearing of the tentorium cerebelli, are distributed amongst these classes as follows :—

(1) Primary uncomplicated breech. Total number of cases, 17; tearing of the tentorium found in 15 (the two exceptions were 134 and 334).

(2) Primary complicated breech. Total number of cases, two; tearing of the tentorium found in neither.

(3) Breech by version. Total number of cases, 28; tearing of the tentorium found in 20. In these 20, the version was performed for the following complications :—

Placenta prævia, 10 cases.

Transverse presentation, five cases (including a case of spontaneous evolution).

Contracted pelvis, three cases.

Brow presentation, two cases.

A broad survey of these figures reveals facts which, to myself at all events, are novel and significant :—(1) Tearing of the tentorium is found in 75 per cent. of dead fœtuses delivered by the breech when all classes of breech delivery are included. (2) Tearing of the tentorium is found in 88 per cent. of dead fœtuses in cases of primary uncomplicated breech presentation, *i.e., as the result of normal breech labour.* In none of these cases was there any reason for undue resistance to the passage of the head, from contraction of the maternal passages or from excessive size or malposition of the fœtal head. Nor was there any apparent reason for rapid delivery of the fœtus. It must be admitted that tearing was favoured by prematurity of the fœtus (for this purpose I include fœtuses below 48 cm. long) in five cases (42, 163, 200, 226 and 314). (3) Tearing of the tentorium is found in 71 per cent. of dead fœtuses delivered after podalic version. This is not so surprising; rapid delivery was no doubt practised in some of the cases of placenta prævia, and in those of transverse presentation complicated by prolapse of the cord; and in the cases of contracted pelvis there was great resistance to the passage of the head. But the significance of these findings is this : it is more than likely that some of these fœtuses escaped death from the primary complication only to meet it as the result of breech delivery. In any case it is a pity to kill a fœtus twice. It is true that in many of these cases the version was performed for the sake of the mother, whose interests must always be paramount; but if a breech delivery be properly managed there should never be sufficient cranial stress to lead to tearing of the tentorium—excluding, of course, cases where rapid delivery is indicated. This aspect of the question is discussed more fully in the section on breech delivery.

On the Actual Causation of Fœtal Death in Cases of Excessive Cranial Stress.

In this condition, as, indeed, in most others, it is impossible to assign the exact cause of death. Quite apart from the two great visible effects of tearing and hæmorrhage, we have to consider the general rise of intracranial pressure, the evidence of which is not seen post mortem. What part this plays in causing death must be left to the imagination; perhaps the crowding down of the medulla oblongata into the foramen magnum results in dangerous pressure on the so-called vital

centres. In cases where there is a large, or moderate, hæmorrhage, it is fair to assume that the fœtus has been killed by the hæmorrhage. But even this does not always kill the fœtus, for I have performed post mortems on several infants who had survived birth by as much as two days, and found extensive bilaterial tears of the tentorium and large hæmorrhages; such infants had, of course, been in a precarious condition since birth. The position of the hæmorrhage is probably of importance. A small hæmorrhage confined beneath the tentorium would be more dangerous than a larger and more diffuse one above this region. The actual tears, unaccompanied by hæmorrhage, cannot be regarded as fatal injuries; the septa of the dura mater are not vital structures, nor are they the source of hæmorrhage. Furthermore, evidence of tearing of the tentorium may be found in infants who have survived birth for a long time. Having survived the original stress, there is no reason why a child should be much the worse for a torn tentorium, provided there has been no, or only very slight, hæmorrhage. It cannot yet be stated what precisely are the effects in after life of these injuries; there is no collected evidence. I feel sure that if pathologists who are in the habit of performing post mortems on children were to look for evidence of these tears, they would find it surprisingly often. I have personally found one such example in a child six months old, in whom there was a large complete tear with healed edges, in the left side of the tentorium. There is, unfortunately, no clinical record of this case, as the child died shortly after admission into hospital, before notes could be made. A point of great interest was the oblique shape of the head which had persisted since birth; the permanent alteration in the shape of the head, in combination with a torn tentorium, is of great importance in throwing light on the aetiology of the rare infantile condition known as "oblique head." It also tends to support my theory that the septa of the dura mater act as supports or stays to the bones.

TABLE IV.

DETAILS OF THE 81 CASES OF TORN TENTORIUM CEREBELLI.

No.	Para	Length.	Presentation.	Complications and operations.	Nature of tear in Tentorium.	Hæmorrhage.	Condition of fœtus at birth.
33	I.	53	Vertex, O.A. -	Contracted pelvis ; forceps -	R, complete ; L, incomplete	Extensive, in all fossæ	Heart beating after birth.
42	II.	43	Primary breech -	Delivery not difficult - -	R, incomplete ; L, complete	Extensive in middle and post. fossæ.	No note.
45	VII.	56	Vertex, L.O.P. -	Forceps ; difficult delivery -	R. complete ; L, complete -	Extensive - -	No note.
51	I.	45	Vertex, R.O.P. -	Manual rotation ; forceps -	R, complete ; L, incomplete	Moderate - - -	Dead at birth.
57	IV.	50	Primary breech -	Delivery not difficult - -	R, incomplete ; L, complete	Extensive in middle and post. fossæ.	No note.
82	VII.	52	Breech by version	Placenta prævia ; bag, followed by version.	R, none ; L, complete -	Moderate - -	Dead at birth.
87	VII.	52	Primary breech -	Delivery not difficult - -	R, complete ; L, complete : falx torn : straight sinus opened.	Extensive, in all fossæ	General œdema of fœtus.
103	I.	40	Vertex - •	Placenta prævia : high forceps	R, complete ; L, complete -	Moderate - - -	Heart beating after birth.
108	I.	52	Vertex, L.O.P. -	Prolapse of cord ; forceps -	R, complete ; L, complete : depressed fracture of L. parietal bone.	Extensive, in all fossæ	Heart beating after birth.
111	VIII.	52	Breech by version	Premature rupture of membranes ; bag : prolapse of arm : version.	R, incomplete ; L, complete	Extensive, in all fossæ	Dead at birth.
135	I.	52	Vertex, O.A. -	Contracted pelvis ; forceps -	R, complete ; L, complete	Extensive, in all fossæ	Heart beating after birth.
142	I.	39	Breech by version	Placenta prævia ; version -	R, complete ; L, incomplete	Slight - - -	Dead at birth.
156	III.	48	Vertex, L.O.P. -	Natural delivery ; second of twins.	R, complete ; L, complete -	Moderate, middle and post. fossæ.	Heart beating after birth.
157	X.	47	Vertex, O.A. -	Prolapse of cord : high forceps	R, complete ; L, complete -	Extensive, in all fossæ	Heart beating after birth
163	VI.	42	Primary breech	Arms extended - - -	R, complete ; L, complete -	Extensive, in middle and post. fossæ.	Not noted.

165	VIII.	48	Breech by version	Placenta prævia, version -	R, incomplete ; L, complete	Extensive - -	Dead at birth.
169	II.	51	Vertex, O.A. -	Prolapse of cord, forceps -	R, incomplete ; L, incomplete.	Slight - - -	Dead at birth.
175	II.	53	Vertex, O.A. -	Contracted pelvis ; pubiotomy : forceps.	R, complete ; L, incomplete	Extensive, in middle and post. fossæ.	No note.
176	V.	46	Vertex, O.A. -	Rapid 2nd stage ; severe perineal tear : former extensive colpoperineorrhaphy	R, none ; L, incomplete -	Extensive, in all fossæ	Fœtal syphilis.
177	I.	48	Primary breech -	Difficult delivery of head -	R, none ; L, complete -	Slight - - -	Heart beating after birth.
179	II.	53	Vertex, O.A. -	Contracted pelvis : forceps -	R, complete ; L, incomplete : falx torn.	None - - -	Dead at birth.
191	V.	51	Vertex, O.A. -	Accidental hæmorrhage : pituitrin.	R, incomplete ; L, incomplete.	Moderate - - -	Dead at birth.
196	V.	48	Vertex, O.A. -	Natural delivery ; second of twins.	R, incomplete ; L, none -	Slight - - -	Dead at birth.
200	I.	40	Primary breech -	Delivery not difficult - -	R, none ; L, incomplete -	Moderate - - -	Dead at birth.
205	IX.	47	Primary breech -	Delivery not difficult - -	R, incomplete ; L, incomplete.	Moderate in middle and post. fossæ.	Heart beating after birth.
209	I.	49	Breech by version	Transverse presentation ; version.	R, none ; L, complete -	Extensive - -	Dead at birth.
212	XI.	47	Vertex, O.A. -	Natural delivery - - -	R, complete ; L, complete -	Extensive, chiefly in middle fossa.	General œdema of fœtus.
220	VI.	51	Brow - -	Conversion into face ; forceps	R, complete ; L, complete : falx torn extensively ; L. lateral sinus opened.	Very extensive - -	Dead at birth.
226	XII.	36	Primary breech -	Arms extended ; difficult delivery.	R, incomplete ; L, incomplete.	Extensive in middle and post. fossæ.	Dead at birth ; liver ruptured.
231	I.	52	Vertex, O.A. -	Contracted pelvis ; forceps followed by Cæsarean section	R, complete ; L, none -	Moderate, in post. fossæ	Heart beating after birth.
236	V.	53	Vertex - -	Placenta prævia : prolapse of cord ; forceps.	R, incomplete ; L, incomplete.	Extensive - -	Dead at birth.
259	VIII.	49	Breech by version	Contracted pelvis ; induction, version.	R, complete ; L, incomplete	Moderate, in middle and post. fossæ.	Dead at birth.
263	VI.	49	Breech by version	Placenta prævia : version -	R, complete ; L, incomplete	None - - -	No note.
265	VI.	47	Vertex - -	Placenta prævia : bag : forceps.	R, complete ; L, complete -	Extensive ; vein of Galen torn.	Dead at birth.

TABLE IV.

No.	Para	Length.	Presentation.	Complications and operations.	Nature of tear in Tentorium.	Hæmorrhage.	Condition of fœtus at birth.
268	I.	48	Breech by version	Transverse presentation ; version.	R, incomplete ; L, incomplete.	Moderate	No note.
269	II.	52	Version	Placenta prævia ; bag : forceps.	R, complete ; L, incomplete	Extensive, in all fossæ	Dead at birth.
272	I.	54	Primary breech	Prolapse of cord ; leg-traction	R, incomplete ; L, incomplete.	Moderate, in middle and post fossæ.	Dead at birth.
283	VI.	55	Primary breech	Difficult delivery of head	R, incomplete ; L, none	Extensive, in all fossæ	No note.
293	IV.	47	Vertex	Accidental hæmorrhage : natural delivery.	R, incomplete ; L, incomplete.	Extensive, in all fossæ	Dead at birth.
301	V.	51	Breech by version	Transverse ; prolapse of cord ; version.	R, complete ; L, incomplete	Moderate	Heart beating after birth.
302	II.	51	Breech by version	Placenta prævia	R, incomplete ; L, incomplete.	None	No note.
303	I.	49	Breech by version	Contracted pelvis ; forceps ; prolapse of cord ; version.	R, complete ; L, complete	Extensive	Dead at birth.
305	I.	48	Breech by version	Brow presentation ; forceps : version.	R, incomplete ; L, incomplete ; falx torn.	Moderate, middle and post. fossæ.	No note.
311	I.	53	Vertex, O.P.	Difficult forceps	R, complete ; L, complete	Extensive, middle and post. fossæ.	Dead at birth.
312	II.	50	Vertex, O.A.	Long 2nd stage ; natural delivery.	R, none ; L, complete	Extensive, middle and post. fossæ.	Heart beating after birth.
314	VIII.	45	Primary breech	Difficult delivery of head	R, complete ; L, complete	Extensive, in all fossæ	Heart beating after birth.
315	V.	48	Vertex	Prolapse of cord ; forceps ; rapid delivery.	R, incomplete ; L, none	Slight	Dead at birth.
317	V.	37	Vertex	Accidental hæmorrhage ; natural delivery.	R, incomplete ; L, incomplete.	None	Heart beating after birth.
337	IV.	32	Transverse	Spontaneous evolution	R, complete ; L, complete	Extensive	No note.
338	X.	46	Vertex	Placenta prævia ; pituitrin	R, incomplete ; L, none	None	No note.

339	X.	51	Breech by version	Brow presentation; version	R, complete ; L, complete	Moderate, middle and post. fossæ.	Heart beating after birth.
340	VIII.	47	Vertex, O.A.	Forceps ; difficult extraction	R, complete ; L, complete	Moderate, middle and post. fossæ.	Heart beating after birth.
341	VI.	45	Vertex	Prolapsed cord ; natural delivery ; second of twins.	R, complete ; L, complete	Extensive	Dead at birth.
343	VII.	41	Breech by version	Placenta prævia ; version	R, complete ; L, complete	Moderate	No note.
344	III.	38	Breech by version	Placenta prævia ; version	R, complete ; L, complete	Moderate	No note.
350	I.	47	Vertex	Contracted pelvis ; forceps	R, incomplete ; L, incomplete.	Moderate	Dead at birth.
353	I.	51	Vertex	Placenta prævia ; bag ; natural delivery.	R, incomplete ; L, complete	Moderate	Dead at birth.
355	I.	49	Vertex, O.P.	Long labour ; manual rotation ; forceps.	R, incomplete ; L, none	Moderate	No note.
356	I.	56	Primary breech	Difficult delivery of head by forceps.	R, complete ; L, complete ; falx torn.	Moderate	Dead at birth.
358	I.	50	Vertex, O.A.	Forceps ; long 2nd stage	R, complete ; L, complete	Extensive	Heart beating after birth.
360	VII.	46	Vertex	Placenta prævia ; bag ; natural delivery.	R, complete ; L, complete	Extensive	No note.
361	I.	47	Vertex, O.A.	Long 2nd stage ; forceps ; first of twins.	R, none ; L, complete	Slight	No note.
363	XI.	52	Primary breech	Easy delivery	R, complete ; L, complete	Moderate, in post. fossa	No note.
364	I.	45	Vertex, O.A.	Natural delivery	R, none ; L, incomplete	Slight	No note.
369	V.	49	Vertex, O.A.	Precipitate labour ; cardiac disease.	R, complete ; L, complete	Moderate	No note.
374	VI.	50	Breech by version	Placenta prævia ; version	R, complete ; L, complete	Extensive	Dead at birth.
386	XI.	52	Breech by version	Contracted pelvis ; prolapse of cord ; version.	R, complete ; L, complete	Extensive in all fossæ	Dead at birth.
390	III.	41	Vertex	Accidental hæmorrhage ; natural delivery.	R, complete ; L, none	Moderate	Heart beating after birth.
397	X.	54	Primary breech	Body born long before arrival of doctor ; delivery of head difficult.	R, complete ; L, complete	Moderate	Dead at birth.
398	I.	50	Vertex	Rapid labour ; induction for albuminuria.	R, complete ; L, incomplete	Extensive	No note.

TABLE IV.

No.	Para	Length.	Presentation.	Complications and operations.	Nature of tear in Tentorium.	Hæmorrhage.	Condition of fœtus at birth.
400	I.	52	Vertex, O.A. -	Natural labour ; cord tightly round neck.	R, none ; L, incomplete -	Moderate - - -	Dead at birth.
401	I.	53	Primary breech	Arms extended - - -	R, complete ; L, complete -	Extensive - -	Heart beating after birth.
404A	VI.	51	Breech by version	Placenta prævia ; version ; one of twins.	R, incomplete ; L, incomplete.	Slight - - -	Dead at birth.
404B	VI.	54	Breech by version	Placenta prævia : version ; one of twins.	R, incomplete ; L, incomplete.	None - - -	Dead at birth.
406	II.	51	Vertex, L.O.P -	Natural delivery - - -	R, complete ; L, none -	Moderate - - -	Dead at birth.
414	VIII.	49	Face - -	Long labour ; natural delivery	R, complete ; L, complete -	Moderate - - -	No note.
415	I.	51	Vertex, O.A. -	Forceps for long 2nd stage -	R, complete ; L, incomplete	Moderate, middle and post. fossæ.	Heart beating after birth.
418	I.	50	Vertex, O.A. -	Contracted pelvis ; forceps ; difficult extraction.	R, complete ; L, incomplete	Extensive, middle and post. fossæ.	Dead at birth.
420	I.	50	Vertex,O.A -	Contracted pelvis ; forceps -	R, complete ; L, incomplete	Slight - - -	Dead at birth.
421	V.	52	Vertex, O.A -	Natural delivery - - -	R, incomplete ; L, incomplete	Moderate, middle and post. fossæ.	Dead at birth.
422	VIII.	55	Vertex, O.P. -	Forceps ; difficult extraction	R, complete ; L, complete -	Extensive, in all fossæ	Dead at birth.

SECTION VI.

Some other morbid fœtal conditions.

(1) Acute Anaërosis.

(2) Cerebral Hæmorrhage other than associated with torn Tentorium Cerebelli.

(3) Fœtal Injuries.

(4) Fœtal Deformities.

VI.—(1) Acute Anaërosis.

A very common post mortem finding in recently dead, mostly fresh, fœtuses is multiple petechial hæmorrhages on the surface of certain organs, especially the lung, heart, liver and thymus: but in extreme cases they may be seen on any organ. In the lung they are beneath the visceral pleura, on the heart beneath the visceral pericardium, in the liver they are in the form of subcapsular hæmorrhages. Occasionally, a subcapsular hæmorrhage of considerable size may be found in the liver. Very often, in association with these, may be found various forms of intracranial hæmorrhage. Usually this takes the form of a fine film of subdural hæmorrhage over the surface of the cerebral hemispheres; sometimes there is a small amount of hæmorrhage at the base of the brain. An extremely common form is between the layers of the falx cerebri, where a hæmatoma is formed (Fig. 13). Still another form consists of small multiple hæmatomata in the pia-arachnoid tissue over the surface of the brain. Occasionally there is a considerable subdural hæmorrhage. On section the organs appear congested, and this appearance is amply confirmed on microscopical section; sometimes even interstitial hæmorrhages are found. These visceral hæmorrhages have been most carefully studied by Dr. Herbert Spencer, who has published his researches in the *Transactions of the Obstetrical Society of London*, 1892, Vol. XXXIII., under the title of "On Visceral Hæmorrhages in Still-Born Children: An Analysis of 130 autopsies—being a contribution to the study of the causation of still-birth." No one interested in fœtal pathology can omit the study of this pioneer piece of work; the main part of the paper consists of a description of the naked-eye and microscopic appearances of the various viscera as regards congestion and hæmorrhage.

Forty-five examples were found amongst the fresh fœtuses and seven amongst the macerated. The essential details of these cases are as follows:—

No. 27.—Accidental hæmorrhage; breech labour, a few petechiæ. Slight hæmorrhage at base of brain.

No. 36.—Accidental hæmorrhage; a few petechiæ, much subdural hæmorrhage over hemispheres and beneath tentorium.

No. 46.—Accidental hæmorrhage; forceps, petechiæ +, subdural hæmorrhage over left cerebral hemisphere, hæmatoma of falx cerebri.

No. 47.—Accidental hæmorrhage, petechiæ +.

No. 64.—Albuminuria of pregnancy, spontaneous labour, petechiæ ++, hæmorrhage between layers of falx cerebri, placenta not examined.

No. 76.—Accidental hæmorrhage, petechiæ +, hæmorrhage between layers of falx cerebri.

No. 136.—Placenta prævia, petechiæ +, hæmorrhage between layers of falx.

No. 140.—Albuminuria of pregnancy, induction, prolapse of cord; petechiæ ++, much subdural hæmorrhage in posterior and middle fossæ.

No. 142.—Placenta prævia. Petechiæ +, internal version, torn tentorium and cerebral hæmorrhage.

No. 143.—Accidental hæmorrhage, petechiæ +, hæmorrhage between layers of falx cerebri.

No. 159.—Accidental hæmorrhage, petechiæ +, hæmorrhage between layers of falx cerebri.

No. 165.—Placenta prævia, internal version, a few petechiæ, torn tentorium and cerebral hæmorrhage.

No. 167.—Accidental hæmorrhage, petechiæ +, large subcapsular hæmorrhage on liver, diffuse slight hæmorrhage around cerebellum, hæmorrhage between layers of falx.

No. 169.—Prolapsed cord, forceps, petechiæ ++, torn tentorium, cerebral hæmorrhage.

No. 191.—Accidental hæmorrhage, petechiæ +, torn tentorium and cerebral hæmorrhage.

No. 194.—Prolapse of cord, petechiæ +, subcapsular hæmorrhages on liver.

No. 195.—Accidental hæmorrhage, petechiæ +, extensive subdural cerebral hæmorrhage.

No. 196.—Twin labour, a few petechiæ, torn tentorium and cerebral hæmorrhage.

No. 203.—Placenta prævia; a few petechiæ, general subdural hæmorrhage over both hemispheres, hæmatoma of falx cerebri.

No. 205.—Breech, born before arrival of attendant, a few petechiæ, torn tentorium and cerebral hæmorrhage.

No. 208.—Prolapse of cord, placenta prævia, petechiæ +, subcapsular hæmorrhage on liver.

No. 216. — Transverse presentation, prolapse of cord, petechiæ +, subcapsular hæmorrhage on liver.

No. 218.—Accidental hæmorrhage, a few petechiæ, no cerebral hæmorrhage.

No. 220.—Brow presentation, forceps, petechiæ, torn tentorium and cerebral hæmorrhage.

No. 235.—Contracted pelvis, induction of labour, a few petechiæ, placenta not examined.

No. 255.—Placenta prævia, a few petechiæ, subtentorial hæmorrhage.

No. 258.—Obstructed labour, perforation of head, petechiæ +, fœtal head not examined.

No. 261.—Placenta prævia, version, petechiæ +.

No. 263.—Placenta prævia, version, a few petechiæ. No cerebral hæmorrhage.

No. 264.—Prolapse of cord. A few petechiæ.

No. 277.—Concealed accidental hæmorrhage, Cæsarean section, petechiæ ++, and a large subcapsular hæmorrhage on liver. Fœtal head not examined.

No. 301.—Transverse presentation, prolapse of cord, version, petechiæ +, torn tentorium and cerebral hæmorrhage.

No. 315.—Prolapse of cord. Forceps, petechiæ +, torn tentorium and cerebral hæmorrhage.

No. 326.—Placenta prævia, petechiæ +, hæmorrhage between layers of falx.

No. 331.—Obstructed labour, craniotomy, petechiæ +.

No. 339.—Brow presentation, delivery by version, petechiæ, torn tentorium and cerebral hæmorrhage.

No. 347.—Vertex presentation, natural delivery, petechiæ ++, slight film of diffuse hæmorrhage over both cerebral hemispheres, placenta not examined; cause of death unknown.

No. 356.—Breech labour, difficulty in delivering after-coming head, forceps, petechiæ +, torn tentorium and cerebral hæmorrhage.

No. 366.—Concealed accidental hæmorrhage, Cæsarean section, petechiæ, slight hæmorrhage over both cerebral hemispheres, hæmatoma of falx.

No. 368.—Accidental hæmorrhage, petechiæ very few, subdural hæmorrhage, hæmatoma of falx.

No. 373.—Placenta prævia, natural vertex delivery. A few petechiæ, much subdural hæmorrhage.

No. 381.—Prolapsed cord, natural vertex delivery, a few petechiæ, slight subdural hæmorrhage.

No. 389.—Albuminuria of pregnancy, petechiæ +, hæmorrhage between layers of falx.

No. 398.—Albuminuria of pregnancy, induction of labour, petechiæ, torn tentorium with cerebral hæmorrhage. Placenta not examined.

No. 418.—Contracted pelvis, forceps, petechiæ ++, torn tentorium.

Amongst macerated fœtuses, petechiæ and hæmorrhage are only likely to be noticed if the maceration is in an early stage. The details of the seven macerated fœtuses are :—

No. 170.—Placenta prævia, central, bag used, a few petechiæ, head not examined.

No. 180.—Placenta prævia, petechiæ +, head not examined.

No. 186.—Syphilis, placenta not examined, petechiæ ++, a few subcapsular hæmorrhages on liver.

No. 192.—Concealed accidental hæmorrhage, petechiæ ++, torn tentorium.

No. 197.—Retroplacental hæmatoma. Hæmorrhage between layers of falx cerebri, petechiæ + +.

No. 228.—Retroplacental hæmatoma and large interstitial placental hæmorrhage. Petechiæ +, subcapsular hæmorrhages on liver.

No. 290.—Probable syphilis. Wassermann +, petechiæ +, placenta healthy.

The details of these cases are given to show two points: (1) these petechiæ and other hæmorrhages in the organs are nearly always accompanied by some form of intracranial hæmorrhage, generally subdural or between the layers of the falx cerebri; occasionally the amount of hæmorrhage is considerable. (Refer to Section IV. (5) on "Cerebral Hæmorrhage, other than that associated with torn tentorium cerebelli.") (2) The type of case in which the condition occurs. There were 17 cases of accidental hæmorrhage, 11 of placenta prævia, and seven of prolapsed cord. Thus, 37 of the 45 cases were associated with extensive and acutely-arising interference with the utero-placental or fœto-placental circulation. "Acute anaërosis" is a better name for it than "acute asphyxia," and I have adopted the former term.

It is interesting to enquire into the reason of the origin of such an intense venous congestion. I offer the following suggestion. When the fœtus begins to suffer from lack of oxygen, it is more than likely that it is stimulated to make the muscular movements of respiration. Recent work has suggested that when this happens there occurs an alteration in the relationship of the thoracic viscera, especially of the heart, which results in kinking or stretching of the ductus arteriosus; it is said that the closure of the ductus arteriosus at the act of respiration after birth is due to this. Should respiratory movements in the fœtus cause a closure, even a partial one, of the ductus arteriosus, about half the blood in the right auricle would be dammed back (*i.e.*, that half which normally would pass through into the right ventricle and thence to the aorta by the ductus arteriosus; the half that passed through the foramen ovale would not, of course, be interfered with). The lungs not being expanded, the circulation of this blood through the lungs would not be permitted. In consequence, with cardiac action still strong, there would arise a most intense venous congestion, due to back pressure from the right auricle.

The discovery in the fœtus of signs of acute anaërosis strongly suggests that the cause of death was separation of the placenta or pressure on the cord.

VI.—(2) Subdural Cerebral Hæmorrhage other than associated with torn Tentorium Cerebelli.

There were 17 cases of this (Nos. 27, 36, 46, 140, 167, 195, 203, 207, 245, 255, 294, 325, 347, 366, 372, 373, 381). *All but five were found in association with signs of acute anaërosis*, and have already been described in that section. From these findings we are justified in concluding there are two chief forms of subdural fœtal cerebral hæmorrhage: (1) that associated with excessive cranial stress, (2) that associated with "acute anaërosis."

The details of these five other cases are the following :—

No. 207.—Eclampsia, natural vertex delivery. No petechiæ seen on any of the organs. There was slight subdural hæmorrhage over the occipital lobe.

No. 245.—Transverse presentation, prolapsed cord, internal version. No petechiæ on fœtal organs. A small hæmatoma was found in both sides of the tentorium cerebelli, near its junction with the falx cerebri; infra-tentorial hæmorrhage, probably due to the rupture of a vessel between the apex of the tentorium and the cerebellum. This observation suggests that the mode of origin of the hæmorrhage was the same as in excessive cranial stress, *i.e.*, the hæmatoma in the tentorium suggests it was stretched just short of the breaking point (*see* p. 55).

No. 294.—Accidental hæmorrhage, version and delivery by breech. No petechiæ found in organs. Hæmorrhage beneath tentorium and over occipital lobes: hæmorrhage probably from sub-tentorial veins. No hæmatoma or tear of tentorium.

No. 325.—Fœtus born before arrival of midwife: the patient and her friends were all drunk. No petechiæ on organs. No lesion of tentorium. Much subdural hæmorrhage and hæmatoma of falx.

No. 372.—Albuminuria of pregnancy. Normal vertex labour. No petechiæ noted in organs. No lesion of tentorium. "Some subdural hæmorrhage."

Of these cases, one (No. 245) is almost certainly due to excessive cranial stress.

I have not examined the brain, in any case, for ventricular hæmorrhage. My interest was concentrated on the tentorium cerebelli and on the fine blood vessels in its neighbourhood, and the method of examining these involved such destruction of the brain as precluded the examination of the ventricles. The leakage of blood into the subdural space, from the ventricles, is a possibility and may account for certain cases of subdural hæmorrhage.

It is probable that ventricular hæmorrhage results from intense engorgement of the fine venules of the choroid plexus, and therefore has a similar mode of origin to subdural hæmorrhage *i.e.* (1) from the general vascular changes that lead to "acute anaërosis," (2) from kinking of the vein of Galen during excessive

cranial stress. I should expect to find ventricular hæmorrhage with either of these states.

VI.—(3) Fœtal Injuries.

Apart from intracranial injuries, only three examples of injuries of the fœtus were found. This is surprising, considering the large proportion of cases of difficult labour in the series.

No. 35.—"Broken neck" Base of skull found completely separated from the spinal column at occipito-atloid joint: spinal cord exposed: prevertebral hæmatoma extending as low as first dorsal vertebra: subdural cerebral hæmorrhage over right parietal and temporal lobes: subtentorial hæmorrhage all around cerebellum and through foramen magnum. Clinically the case was one of obstructed labour due to abnormal enlargement caused by double cystic fœtal kidneys. Head presentation.

No. 226.—Rupture of liver. Premature fœtus, length 36 cm. XII. para. Breech labour; difficulty in bringing down arms. Much blood and clot in peritoneal cavity: the ruptured areas were on the lower anterior surfaces of both lobes. Also torn tentorium and cerebral hæmorrhage.

No. 393. Rupture of liver. Premature fœtus, length 36 cm., transverse presentation, delivered by breech. Liver ruptured on inferior surface, many interstitial and subcapsular hæmorrhages; hæmorrhage into peritoneal cavity.

VI.—(4) Deformities of the Fœtus.

There were 15 examples of this.

Fresh Fœtuses.—General œdema, Nos. 87, 212, 365, 395. Double cystic kidneys, No. 35. Fœtal ascites, No. 75. Anencephaly, No. 188, 206. Kidneys remarkably small, No. 232.

Macerated Fœtuses.—Hydrocephalus, spina bifida, double talipes, No. 5. Spina bifida, double talipes of hands and feet, No. 44. Double talipes, Nos. 227, 328. Hydrocephalus, No. 60. Anencephaly, No. 275.

The cases showing absence of one unbilical and hypogastric artery are placed in Section VIII. (3).

SECTION VII.

Fœtal Weights and Measurements.

(1) Weight-ratios of the Fœtal Organs.

(2) Table V., showing Frequency-distribution of the Various Organs amongst Fœtuses of Different Weights.

(3) Analysis of Table V., and Lists of Cases with Exceptionally Heavy or Light Organs.

(4) The Length-weight-ratio of the Fœtus.

(5) Table VI., showing Distribution of Weight-length-ratios amongst Fœtuses of Different Weights.

VII.—(1) Weight-ratios of the Fœtal Organs.

The number of cases is too small for precise statistical work; such figures are badly wanted.

The large organs have been weighed to the nearest gramme, and the small ones, such as the suprarenal and thyroid, to the nearest half-gramme.

The weights of the fœtal organs are so dependent on the weight of the fœtus (and in this series the fœtuses vary from 600 to over 4,000 grms.) that the employment of the actual weights of the organs is useless for studying means and variations; the ratio of the weight of the organ to the weight of its fœtus must be employed. This ratio is got by dividing the weight of the fœtus by the weight of the organ; a high ratio means a light and a low ratio a heavy organ relative to the weight of the fœtus. The ratios of the organs will be found in Table XIII. The ratios of each important organ have been arranged in Table V. (p. 83), showing their frequency-distribution amongst fœtuses of different weights. In this Table the fœtuses have been grouped into weights differing by 400 grms., and it will also be noticed that the ratios for each organ have been distributed into a convenient number of groups.

A glance at the tables shows how greatly the ratios vary in fœtuses of the same weight. With so much variation the figures are too small to establish a reliable mean for the organs, but the means have nevertheless been calculated, and may be accepted for what they are worth. Attention is at once drawn to the difference in the ratios between fresh and macerated fœtuses. The organs of the former are, in the case of all the organs, heavier than those of the latter: this is especially noticeable in the case of an organ which, like the liver, is richly endowed with enzymes. The difference must be due to the loss of weight by autolysis during the intrauterine retention of the dead fœtus;

the products of autolysis escape through the capsule of the organ and no doubt contribute to the sanguineous fluid found in the body-cavities of the macerated fœtus; the difference may be said to depend on the degree of maceration.

It follows that the organ weights of a macerated fœtus are unreliable and do not represent the true weight of the organ during life.

The display of the organs in frequency-groups gives a rough idea of the mean weight, and shows the most likely weight (the mode), of an organ for a fœtus of a particular weight. It also enables one to pick out the variants at either end of the scale, *i.e.*, the exceptionally heavy or the exceptionally light organs, and such cases can then be studied to see if they throw any light on fœtal pathology.

It should be noted that syphilitic (spirochæte-positive) fœtuses have been placed in a separate group, both because in these fœtuses certain organs are notably increased in weight and because it is desirable to find out, by noting how often or how seldom such increased weight occurs in non-syphilitic fœtuses, if such increase in weight of the organs of syphilitic fœtuses can be accepted as a reliable aid in the diagnosis of fœtal syphilis. This particular point will be found fully dealt with in the syphilitic section.

I shall now study the ratios and draw what conclusions are possible or permissible from such a small array of cases.

The Liver.

The frequency-distributions of the ratios are shown in the tables, which demonstrate how much more irregular is the distribution amongst macerated than amongst fresh fœtuses.

The mean ratio for 162 fresh fœtuses is 21, and for 95 macerated is 35. The difference in the means between fresh and macerated is a striking one and, accepting the view that the organs of macerated fœtuses lose weight by *post mortem* autolysis, is just what would be expected in an organ so rich in intracellular enzymes as the liver.

If the fresh fœtuses are divided into three weight-groups, it will be seen that the ratio is hardly at all influenced by the weight of the fœtus.

600–1,800 grms., 27 fœtuses; mean ratio, 21.
1,800–3,000 grms., 67 fœtuses; mean ratio, 21.
over 3,000 grms., 68 fœtuses; mean ratio, 20.

Compare now these non-syphilitic with syphilitic (spirochæte-positive) fœtuses—

macerated syphilitic, 35 fœtuses, mean ratio, 21 (instead of 35).
fresh syphilitic, 2 fœtuses, mean ratio, 16 (instead of 21).

Those fœtuses (other than syphilitic) whose ratios show an extreme deviation from the mean may now be selected, and their

details examined to find out if there is any relation between exceptionally heavy or light livers and pathological states, maternal or fœtal.

1. *Heavy Livers.*—Any fresh fœtus with a ratio of less than 15 and any macerated fœtus with a ratio of less than 20, has an exceptionally heavy liver. Amongst the 162 fresh fœtuses there are six (Nos. 42, 143, 295, 346, 356 and 422), and amongst the 92 macerated there are fourteen (Nos. 105, 152, 164, 190, 234, 247, 271, 290, 309, 313, 319, 335, 370, and 382) with exceptionally heavy livers.

If the details of the seven fresh fœtuses are examined, the only thing of interest that emerges is that there is one case of undoubted syphilis (No. 229); there was one case of accidental hæmorrhage, one of placenta prævia, two cases of torn tentorium, and, in one (No. 422) the liver contained a large cyst; in one fœtus (No. 143) the thymus and spleen were enlarged, in one (No. 295) the thymus and kidneys were enlarged, and in one (No. 422) the kidneys were exceptionally small. Amongst the fresh fœtuses there seems to be no correlation between enlarged liver and other pathological states.

If now the details of the macerated fœtuses are examined the results are interesting.

No. 105.—Albuminuria of pregnancy, spleen +, suprarenal +, kidneys —, thymus —.
No. 152.—Hydramnion; spleen +, kidneys +.
No. 164.—Wassermann +. Cause unknown.
No. 190.—Albuminuria of pregnancy; placenta +, spleen +.
No. 234.—Wassermann +, spleen +, suprarenals +, kidneys + : "probable syphilis."
No. 247.—Chronic renal disease; spleen +, thymus +.
No. 271.—Placenta prævia.
No. 290.—Wassermann +, placenta +, spleen +, suprarenals +, kidneys + : "probable syphilis."
No. 309.—Chronic renal disease.
No. 313.—Wassermann +, spleen +; classed as "certain syphilis."
No. 319.—Cause of death unknown; suprarenals +, kidneys +.
No. 335.—Accidental hæmorrhage; placenta + : "probable syphilis."
No. 370.—Albuminuria of pregnancy.
No. 382.—Albuminuria of pregnancy; thymus +, kidneys +.

A study of this list reveals the fact that one half are cases associated with albuminuria of pregnancy in the mother, viz., four cases of albuminuria of pregnancy, two of chronic renal disease, and one of accidental hæmorrhage; also that there are three cases of "probable" and one of "certain" syphilis. These two revelations should assist in placing in their proper category the two cases of "cause of death unknown."

As to the association of enlarged liver with enlargement of other organs, it will be noticed that in half the cases the spleen was enlarged (including two cases of "probable" and one of "certain" syphilis).

2. *Light Livers.*—Any fresh fœtus with a ratio of more than 30 has an exceptionally light liver. There are five such (Nos. 13, 159, 194, 209, and 225); an examination of the tables reveals that two of them are cases of accidental hæmorrhage and one of extensive infarction of the placenta, but nothing else of interest.

In the case of macerated fœtuses, the great loss of liver weight by post mortem autolysis makes it difficult to select cases in which the liver was exceptionally light during the life of the fœtus. But it is justifiable to assume that livers with an extremely high ratio possess a degree of lightness more than dependent on autolysis. It is here assumed that macerated fœtuses have an exceptionally light liver when the ratio exceeds 55. There are 10 such fœtuses (Nos. 30, 122, 210, 224, 230, 270, 323, 357, 359, and 394). Amongst these there is one case of albuminuria of pregnancy, one case of retroplacental clot and one of "probable syphilis"; in the remainder the cause of death is unknown.

The Spleen.

In the tables showing frequency-distribution the ratios are grouped according to a difference of 50; their distribution is very irregular, especially amongst the macerated fœtuses.

The mean ratio for 160 fresh fœtuses is 360. If the mean of fœtuses of different weights be calculated, it is found that the spleen is relatively lighter in premature than in mature fœtuses—

600–1,800 grms., 27 fœtuses; mean ratio, 390.
1,800–3,000 grms., 66 fœtuses; mean ratio, 339.
over 3,000 grms., 67 fœtuses; mean ratio, 369.

The mean ratio for 93 macerated fœtuses is 481. The difference between fresh and macerated fœtuses is again noticeable, but is not nearly so great as in the case of the liver; this is only to be expected when the ferment content, and the consequent rate and extent of autolysis, of the two organs is compared.

Contrast now these ratios with the ratios for syphilitic fœtuses; in 37 of these, both macerated and fresh, the mean ratio is 171. The ratio is more than twice as high; the heavy spleen of fœtal syphilis is noteworthy.

Extreme deviations from the mean may now be taken, and the cases examined to see if there is any correlation between exceptional spleens and pathological conditions of the fœtus or mother.

1. *Heavy Spleens.*—Spleens with a ratio of less than 200 in fresh fœtuses and with a ratio of less than 300 in macerated fœtuses must be considered exceptionally heavy. There are 11 fresh fœtuses with such a ratio (Nos. 87, 143, 145, 204, 229,

245, 294, 295, 350, 355 and 365). The largest spleen is from a case of "certain" syphilis (No. 229), and the two next largest are from cases of general œdema of the fœtus; the others are divided amongst the usual complications of pregnancy and labour, such as antepartum hæmorrhage, transverse presentation, contracted pelvis, and call for no comment.

In fresh fœtuses enlargement of the spleen does not seem to be especially associated with alterations in the size of other organs.

The macerated fætuses with ratios of less than 300 number 18 (Nos. 5, 34, 65, 68, 105, 110, 152, 168, 190, 197, 211, 234, 247, 290, 313, 362, 384, and 403*b*). Enlargement of the spleen, as is the case with other organs, is more definitely associated with pathological conditions in macerated than in fresh fœtuses. All but three fall into three groups :—

(1) Toxæmia of pregnancy, comprising six cases of albuminuria of pregnancy and one of accidental hæmorrhage.

(2) Two cases of "certain syphilis" and two of "probable syphilis"; in two others the maternal Wassermann reaction was positive, but there were no other signs of fœtal syphilis.

(3) Oedematous state of the ovum, two cases; one of hydrocephalus and one of hydramnion. Note that the two heaviest spleens amongst the fresh fœtuses were from cases of general œdema.

Of the remaining cases, in one the hypogastric and umbilical arteries of one side were absent and in the other two the cause of death is unknown; from the foregoing considerations, the cause was very likely either syphilis or maternal toxæmia.

There is no constant association between enlargement of the spleen and unusual size of any other organ or organs; but it is worthy of note that seven of the 18 cases were associated with an exceptionally heavy liver (three cases of albuminuria of pregnancy, three of probable syphilis, and one of hydramnion).

2. *Light Spleens.*—For fresh fœtuses a ratio of over 600, and for macerated fœtuses a ratio of over 700, are taken as standards of lightness. There are five fresh fœtuses with a ratio of over 600 (Nos. 17, 179, 383, 404, and 421), and it is probably only a coincidence that three of these are cases of placenta prævia.

There are thirteen macerated fœtuses with exceptionally light spleens (Nos. 30, 32, 150, 198, 217, 228, 257, 270, 297, 321, 323, 357, 359); the weights are not reliable, owing to the impossibility of knowing how much weight was lost by autolysis. On looking through the table it will be noticed that three of them are from cases of albuminuria of pregnancy.

The Kidneys.

In the frequency-distribution tables the ratios are placed in groups differing by 10. The distribution of the ratios is very

irregular, for fresh and macerated fœtuses alike. The mean ratio for 162 fresh fœtuses is 123, and for 91 macerated is 149. The difference between fresh and macerated fœtuses is much less than in any other organ. The kidney is the best preserved of all the organs of a macerated fœtus, and undergoes only a slight degree of autolysis after death. By comparing the ratios of fresh fœtuses of different weights, it is seen that there is very little difference between mature and premature fœtuses.

600–1,800 grms., 27 fœtuses; mean ratio, 127.
1,800–3,000 grms., 68 fœtuses; mean ratio, 115.
over 3,000 grms., 67 fœtuses; mean ratio, 130.

(In calculating the mean for the fresh fœtuses, No. 232 has not been included, as the ratio was such a remarkably high one).

The mean ratio for the 37 spirochæte-positive fœtuses is 105. comparison with the ratios for non-syphilitic fœtuses shows that there is a slight increase of the weight of the kidneys in syphilis.

Extreme deviations from the mean may now be considered.

1. *Heavy Kidneys.*—Ratios of below 70, 80 and 90 may be taken as representing exceptionally heavy kidneys for fresh fœtuses of under 1,800 grms., 1,800–3,000 grms., and over 3,000 grms. respectively.

There are 15 such fresh fœtuses with these low ratios (Nos. 47, 57, 75, 177, 187, 226, 252, 259, 295, 368, 374, 383, 386, 400 and 410).

For macerated fœtuses all kidneys may be considered heavy whose ratios are below 90, 100 and 110, for the same fœtal weight-groups respectively. The number of these is 10 (Nos. 5, 110, 152, 201, 234, 267, 290, 319, 362 and 382). Amongst the fresh fœtuses there are two cases of accidental hæmorrhage and three of placenta prævia; the usual accidents of labour are distributed amongst the rest.

Amongst the macerated fœtuses are four cases of albuminuria of pregnancy, two of probable syphilis, and two of œdematous states of the ovum, *i.e.*, hydrocephalus and hydramnion. The albuminurias and probable syphilis cases, and the hydramnion and hydrocephalus, are combined with enlarged liver. The only interesting feature of this analysis is the predominence of pregnancy toxæmias (albuminuria and accidental hæmorrhage) amongst the group of fœtuses with enlarged kidneys.

2. *Light Kidneys.*—The frequency-distribution tables indicate that in fresh fœtuses of under 1,800 grms. weight, kidneys with a ratio of above 160, and in fœtuses over 1,800 grms. weight, those with a ratio of above 170, should be classed as exceptionally light. In macerated fœtuses a ratio of over 190 is indicated.

There are 16 fresh fœtuses (Nos. 28, 87, 135, 157, 203, 212, 232, 248, 288, 302, 303, 305, 315, 337, 395, and 422), and 18 macerated (Nos. 18, 30, 49, 60, 105, 122, 150, 170, 197, 238, 251, 273, 278, 292, 323, 336, 388 and 412).

Examination of the details of these fœtuses does not enable any generalizations to be made, but there are a few interesting cases. All the cases of general œdema of the fœtus are included (and not because the increased body-weight has made the ratio large, because in only one of them is the fœtus excessively heavy in relation to its length); there is one case where both kidneys were exceptionally small (No. 232, with a ratio of 776) and one case where the kidney of the one side was remarkably small (No. 203); two cases of eclampsia where the kidneys showed, histologically, massive coagulative necrosis; and two cases where the hypogastric and umbilical arteries of one side were missing.

The Suprarenals.

In the frequency-distribution tables the ratios are grouped in differences of 50. Their distribution is very irregular, though not so irregular as the spleen ratios.

For 160 fresh fœtuses, the mean ratio works out at 402. The suprarenal is relatively heavier in premature than in mature fœtuses.

600–1,800 grms., 27 fœtuses; mean ratio, 346.
1,800–3,000 grms., 67 fœtuses; mean ratio, 417.
over 3,000 grms., 66 fœtuses; mean ratio, 410.

In macerated fœtuses the mean ratio is higher, undoubtedly for the same reason as in the case of the other organs. For 90 macerated fœtuses the mean ratio is 482.

In the syphilitic fœtus the suprarenal is heavier than in the non-syphilitic. In 35 spirochæte-positive the mean ratio is 364.

Exceptional deviations from the mean may now be taken.

1. *Heavy Suprarenals.*—The frequency-distribution tables indicate that for fresh fœtuses a ratio of under 200 for fœtuses under 1,800 grms. in weight, and a ratio of under 250 for those over 1,800 grms. is indicated. For macerated fœtuses a ratio of under 250 for fœtuses of under 1,800 grms., and a ratio of under 300 for fœtuses of over 1,800 grms. is indicated.

The fresh fœtuses with these ratios number fourteen (Nos. 103, 111, 160, 169, 187, 204, 268, 339, 355, 365, 373, 374, 383, and 404*b*), and the macerated fœtuses ten (Nos. 105, 189, 234, 290, 319, 362, 384, 394, 403*b*, and 407).

Examination of the details of the fresh fœtuses shows that seven were cases of placenta prævia, the remainder were cases of the usual complications of labour. The association of such a high proportion of cases of placenta prævia may be only a coincidence; the explanation of such an association is not clear.

Amongst the macerated fœtuses were three cases of albuminuria of pregnancy, two of probable syphilis and four cases in which the cause of death was undetermined; five of the ten were combined with an enlarged spleen (two albuminurias, two probable syphilis, one unknown).

2. *Light Suprarenals.*—The frequency-distribution tables indicate that the suprarenal is exceptionally light when the ratio is 600 or more in fresh, and 700 or more in macerated fœtuses. There are fourteen fresh (Nos. 13, 28, 75, 82, 87, 107, 116, 135, 137, 188, 209, 212, 301, and 421) and twelve macerated (Nos. 30, 34, 49, 60, 122, 168, 215, 221, 230, 238, 257 and 273) with such ratios. These fœtuses reveal little else of associated pathological interest. Amongst the fresh fœtuses are two with general œdema, and by far the lightest suprarenal is possessed by an anencephalic fœtus (No. 188). Amongst the macerated fœtuses are eight in which the cause of death is " unknown."

The Thymus.

In the frequency-distribution tables the ratios are grouped in differences of 50; the distribution is irregular, the extremes being chiefly in the direction of high ratios. The mean ratio for 159 fresh fœtuses is 393; if the fœtuses are divided into three weight-groups it is seen that the ratio is hardly at all influenced by the fœtal weight, showing that the thymus has the same relative weight both in mature and premature fœtuses.

600–1,800 grms., 26 fœtuses; mean ratio, 412.
1,800–3,000 grms., 67 fœtuses; mean ratio, 387.
3,000 grms. and more, 66 fœtuses; mean ratio, 390.

The mean ratio for 89 macerated fœtuses in 648, pointing to a considerable loss of weight by post mortem autolysis.

Syphilis leads to only a slight increase in weight; the mean ratio for 34 spirochæte-positive fœtuses is 526.

The cases which show an exceptional deviation from the mean may now be taken.

1. *Heavy Thymus.*—Amongst the fresh fœtuses, the frequency-distribution tables indicate ratios of under 200; of these there are nineteen (Nos. 51, 75, 116, 136, 143, 159, 187, 188, 206, 265, 295, 302, 339, 340, 343, 344, 349, 355, 406). Two of the fœtuses are anencephalic, one is a case of fœtal ascites; nine are cases of placenta prævia, two of accidental hæmorrhage, and the remainder are cases with common forms of dystocia.

An abnormally heavy thymus is not particularly associated with abnormal weights of any other organ or organs.

Amongst the macerated fœtuses a ratio of less than 300 may be taken as indicating exceptional size; of such there are nine fœtuses (Nos. 168, 170, 247, 278, 281, 282, 308, 367 and 382). These, again, show no particular association with maternal or fœtal abnormalities or with exceptional weight of other organs.

Syphilis is not a cause of enlarged thymus, as only two syphilitic fœtuses have it (Nos. 296 and 413).

2. *Light Thymus.*—For fresh fœtuses a ratio of 800 or over is indicated. There are nine (Nos. 35, 48, 87, 298, 305, 334, 365, 395 and 419). Amongst these are the three cases of general œdema.

For macerated fœtuses those with a ratio of 1,200 or over may be selected. There are eight of these (Nos. 5, 38, 105, 198, 262, 280, 297, 359); seven are associated with albuminuric states of the mother, *i.e.*, four cases of albuminuria of pregnancy, two of accidental hæmorrhage, and one of chronic renal disease; the other is a case of hydrocephalus.

VII.—(2) TABLE V.

TABLES showing FREQUENCY-DISTRIBUTION of the RATIOS of the VARIOUS ORGANS amongst FŒTUSES of different WEIGHTS.

For each organ there are three Tables :—

I.—Fresh spirochæte-free fœtuses.
II.—Macerated spirochæte-free fœtuses.
III.—Spirochæte-positive fœtuses.

LIVERS.

I.—FRESH NON-SPIROCHÆTE.

Weight of Fœtus.	Ratios.												Number of Fœtuses.
	5	10	15	20	25	30	35	40	45	50	55	60	
600	—	—	—	—	—	—	—	—	—	—	—	—	—
1,000	—	2	4	8	2	—	—	—	—	—	—	—	16
1,400	—	1	2	6	1	—	—	1	—	—	—	—	11
1,800	—	1	7	10	2	—	1	—	—	—	—	—	21
2,200	—	—	10	9	3	—	—	—	—	—	—	—	22
2,600	—	—	7	12	4	1	—	—	—	—	—	—	24
3,000	—	—	19	12	6	—	—	—	—	—	—	—	37
3,400	—	2	4	8	3	1	—	—	—	—	—	—	18
3,800	—	—	1	4	1	—	1	—	—	—	—	—	7
4,200	—	1	2	3	—	—	—	—	—	—	—	—	6
	—	7	56	72	22	2	2	1	—	—	—		162

II.—MACERATED NON-SPIROCHÆTE.

600	—	—	—	—	1	—	—	1	1	2	—	—	5
1,000	1	1	—	3	1	1	3	3	2	1	1	2	19
1,400	—	—	2	—	2	1	1	—	1	1	—	1	9
1,800	—	—	4	—	2	2	—	1	—	2	2	3	16
2,200	—	1	2	6	3	3	1	—	3	—	—	—	19
2,600	—	—	2	1	1	1	2	3	1	—	—	—	11
3,000	—	—	—	2	1	—	2	—	—	—	—	—	5
3,400	—	—	1	1	2	—	1	—	—	1	—	—	6
3,800	—	—	—	—	2	—	—	—	—	—	—	1	3
4,200	—	—	—	1	—	—	1	—	—	—	—	—	2
	1	2	11	14	15	8	11	8	8	7	3	7	95

III.—Spirochæte-positive.

Weight of Fœtus.	Ratios.												Number of Fœtuses.
	5	10	15	20	25	30	35	40	45	50	55	60	
600	—	—	1	1	2	—	—	—	—	—	—	—	4
1,000	2	4	1	—	1	4	—	1	—	—	—	—	13
1,400	—	—	2	2	—	—	—	—	—	—	—	—	4
1,800	—	3	2	1	1	—	1	—	—	—	—	—	8
2,200	—	—	1	1	2	—	—	—	—	—	—	—	4
2,600	—	—	1	1	—	1	—	—	—	—	—	—	3
3,000	1	—	—	—	—	—	—	—	—	—	—	—	1
	3	7	8	6	6	5	1	1	—	—	—	—	37

SPLEENS.

I.—Fresh, non-Spirochæte.

Weight of Fœtus.	Ratios.															Number of Fœtuses.
	50	100	150	200	250	300	350	400	450	500	550	600	650	700	750	
600	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	—
1,000	1	1	1	1	1	1	2	3	1	1	2	–	–	–	1	16
1,400	–	–	–	1	1	1	2	2	1	1	2	–	–	–	–	11
1,800	–	–	2	4	5	1	3	1	2	3	–	–	1	–	–	22
2,200	–	–	3	4	4	4	4	–	1	–	1	–	–	–	–	21
2,600	–	–	1	1	5	6	4	2	2	1	1	–	–	–	–	23
3,000	–	1	1	4	7	6	4	8	2	2	–	2	–	–	–	37
3,400	–	–	–	1	3	6	2	2	1	1	1	–	–	–	–	17
3,800	–	–	–	–	–	2	1	2	1	1	–	–	–	–	–	7
4,200	–	–	–	–	2	1	1	–	–	1	–	1	–	–	–	6
	1	2	8	16	28	28	23	20	11	11	7	3	1	–	–	160

II.—Macerated non-Spirochæte.

Weight of Fœtus.	Ratio.																Number of Fœtuses.
	50	100	150	200	250	300	350	400	450	500	550	600	650	700	750	800	
600	–	–	–	–	–	1	1	–	1	–	–	–	–	–	1	1	5
1,000	–	1	1	1	–	1	1	1	3	2	3	1	2	2	–	1	20
1,400	–	–	1	–	–	–	1	1	–	2	1	1	–	–	–	2	9
1,800	–	–	1	–	2	2	1	–	2	3	1	1	–	–	–	3	16
2,200	1	–		1	1	4	1	1	2	–	2	1	–	1	–	–	15
2,600	–	1	–	1	3	3	2	1	1	1	–	–	–	–	–	1	14
3,000	–	–	–	1	–	–	–	1	1	–	–	–	–	–	–	1	4
3,400	–	–	–	1	1	1	–	–	1	1	1	–	–	–	–	–	6
3,800	–	–	–	–	–	–	–	–	–	1	–	–	–	–	–	1	2
4,200	–	–	–	–	–	–	–	–	–	1	–	–	1	–	–	–	2
	1	2	3	5	7	12	7	5	11	11	8	4	3	3	1	10	93

III.—Spirochæte-positive.

Weight of Fœtus.	Ratio.																Number of Fœtuses.
	50	100	150	200	250	300	350	400	450	500	550	600	650	700	750	800	
600	–	1	2	1	–	–	–	–	–	–	–	–	–	–	–	–	4
1,000	2	7	–	–	2	–	1	–	–	–	1	–	–	–	–	–	13
1,400	–	2	2	1	–	–	–	–	–	–	–	–	–	–	–	–	5
1,800	4	2	1	–	–	–	–	–	–	–	–	–	–	–	–	–	7
2,200	1	1	–	1	1	–	–	–	–	–	–	–	–	–	–	–	4
2,600	–	–	–	1	1	–	1	–	–	–	–	–	–	–	–	–	3
3,000	1	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1
	8	13	5	4	4	–	2	–	–	–	1	–	–	–	–	–	37

KIDNEYS.

I.—Fresh non-Spirochæte.

Weight of Fœtus.	Ratio.														Number of Fœtuses.
	50	60	70	80	90	100	110	120	130	140	150	160	170	180	
600	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
1,000	1	3	2	1	4	1	–	3	1	–	–	–	–	–	16
1,400	–	–	1	–	1	2	3	–	–	1	–	2	–	1	11
1,800	–	1	–	2	3	6	1	3	2	–	3	–	–	1	22
2,200	1	1	–	3	3	3	–	2	5	1	1	1	–	1	22
2,600	–	2	–	1	2	6	1	4	2	2	3	–	1	–	24
3,000	–	–	3	–	2	7	3	5	7	2	3	2	2	1	37
3,400	–	–	–	3	1	1	2	4	3	1	–	–	–	3	18
3,800	–	–	–	–	–	–		1	–	2	2	–	–	1	6
4,200	–	–		–	–	1	1	1	–	1	–	–	–	2	6
	2	7	6	10	16	27	11	23	20	10	12	5	3	10	162

II.—Macerated non-Spirochæte.

600	–	–	–		–	1	1	1	–	–	–	–	2	–	5
1,000	–	–	1	1	2	1	1	3	1	2	1	1	–	4	18
1,400	–	–	–	1	1	–	–	2	1	–	–	–		4	9
1,800	–	–	1	–	–	2	1	1	–	–	3	–	1	5	14
2,220	–	–	1	–	2	4	4	–	3	2	1	1	–	–	18
2,600	–	–	–	1	–	–	2	1	–	1	–	1	1	4	11
3,000	–	–	–	–	–	–	–	1	1	–	–	1	–	2	5
3,400	–	–	1	–	–	1	1	–	1	–	–	–	–	2	6
3,800	–	–	–	–	–	–	–	1	–	–	–	–	–	2	3
4,200	–	–	–	–	–	–	–	–	–	–	–	–	–	2	2
	–	–	4	3	5	9	10	10	7	5	5	4	4	25	91

III.—Spirochæte-positive.

Weight of Fœtus.	Ratio.														Number of Fœtuses.
	50	60	70	80	90	100	110	120	130	140	150	160	170	180	
600	–	–	–	1	1	1	1	–	–	–	–	–	–	–	4
1,000	1	2	2	1	2	–	1	–	–	–	1	–	3	–	13
1,400	–	–	–	2	–	1	–	1	–	–	–	–	–	–	4
1,800	–	–	4	–	–	–	–	3	1	–	–	–	–	–	8
2,200	–	–	–	–	–	–	1	2	–	1	–	–	–	–	4
2,600	1	–	–	–	1	–	1	–	–	–	–	–	–	–	3
3,000	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1
	2	2	6	4	4	3	4	6	1	1	1	–	3	–	37

SUPRARENALS.

I.—Fresh Spirochæte-free.

Weight of Fœtus.	Ratio																	Number of Fœtuses.
	100	150	200	250	300	350	400	450	500	550	600	650	700	750	800	850	900	
600	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
1,000	–	3	4	4	3	1	1	–	–	–	–	–	–	–	–	–	–	16
1,400	–	–	–	2	2	2	1	–	3	–	–	–	–	–	–	–	1	11
1,800	–	–	1	3	5	5	3	–	2	–	1	–	–	–	–	–	1	21
2,200	–	1	2	1	4	4	2	3	2	1	–	–	–	–	–	–	1	22
2,600	–	1	1	3	6	4	3	1	2	2	1	–	–	–	–	–	1	24
3,000	–	–	1	8	9	8	3	2	–	–	3	–	–	1	–	–	–	35
3,400	–	–	2	1	3	5	2	2	–	1	1	–	–	1	–	–	–	18
3,800	–	–	–	–	–	–	–	2	4	–	1	–	–	–	–	–	–	7
4,200	–	–	1	–	–	–	1	1	1	1	–	–	–	1	–	–	–	6
	–	5	12	22	32	29	16	11	14	5	7	–	–	3	–	–	4	160

II.—Macerated Spirochæte-free.

600	–	–	–	1	1	1	–	–	–	–	–	–	–	–	–	1	–	4
1,000	–	–	2	3	2	–	4	1	2	3	–	1	–	–	–	–	–	18
1,400	–	–	1	1	2	1	1	–	2	–	–	–	1	–	–	–	–	9
1,800	–	–	–	1	1	–	2	4	–	–	4	1	–	–	1	–	–	14
2,200	–	–	3	1	2	4	1	1	1	2	–	–	–	–	–	2	1	18
2,600	–	–	1	–	–	2	1	–	2	–	1	2	1	–	–	1	–	11
3,000	–	–	–	–	–	2	–	1	1	–	1	–	–	–	–	–	–	5
3,400	–	1	–	–	1	1	–	1	1	1	–	–	–	–	–	–	–	6
3,800	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	2	3
4,200	–	–	–	–	–	–	–	–	–	–	–	–	1	1	–	–	–	2
	–	1	7	7	10	11	9	8	9	6	6	4	3	1	1	4	3	90

III.—Spirochæte-positive.

600	–	1	–	1	1	–	–	–	1	–	–	–	–	–	–	–	–	4
1,000	–	3	3	–	1	1	3	1	1	–	–	–	–	–	–	–	–	13
1,400	–	–	–	–	–	2	–	1	1	–	–	–	–	–	–	–	–	4
1,800	–	–	–	2	1	1	–	1	1	–	–	–	–	–	–	–	–	6
2,200	–	–	–	–	1	–	2	1	–	–	–	–	–	–	–	–	–	4
2,600	–	–	–	–	1	1	1	–	–	–	–	–	–	–	–	–	–	3
3,000	–	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	1
	–	4	3	3	5	5	6	4	5	–	–	–	–	–	–	–	–	35

THYMUS.

I.—Fresh.

Weight of Fœtus.	Ratio																	Number of Fœtuses.
	100	105	200	250	300	350	400	450	500	550	600	650	700	750	800	850	900	
600	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
1,000	2	–	4	2	2	1	–	–	1	–	–	1	–	–	1	–	1	15
1,400	–	1	2	2	1	–	3	1	–	–	–	1	–	–	–	–		11
1,800	2	2	5	4	–	–	3	1	2	–	–	–	2	–	–	–	1	22
2,200	1	1	10	1	2	1	–	–	3	1	–	–	–	–	–	1	1	22
2,600	1	5	3	5	2	2	–	1	1	1	–	–	1	–	1	–	–	23
3,000	–	1	10	5	5	2	2	3	2	2	1	1	–	–	–	–	1	35
3,400	–	2	2	1	4	5	–	1	–	1	1	–	–	–	–	1		18
3,800	–	–	2	–	–	3	1	–	–	–	–	–	–	–	–	–	1	7
4,200	–	1	–	2	1	1	1	–	–	–	–	–	–	–	–	–	–	6
	6	13	38	22	17	15	10	7	9	5	2	3	3	–	2	2	5	159

II.—Macerated.

600	–	–	–	1	–	–	–	–	–	1	–	–	–		–	–	2	4
1,000	–	–	1	–	–	1	2	–	1	1	1	2	1	2	–	2	3	17
1,400	–	–	–	1	–	1		1	–	–	–	–	1	2	–	–	3	9
1,800	–	–	–	2	3	2	1	1	–	–	1	–	1	1	1	–	2	15
2,200	–	–	–	1	4	2	2	2	1	–	1	2	–	1	1	–	2	19
2,600	–	–	–	2	–	2	–	1	1	–	–	–	2	–	–	–	1	9
3,000	–	–	–	–	1	–	2	1	–	–	–	–	–	–	1	–	–	5
3,400	–	–	1	–	1	2	–	–	1	–	–	–	–	–	–	–	1	6
3,800	–	–	–	–	–	–	–	–	–	–	2	–	–	–	–	–	1	3
4,200	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	1	1	2
	–	–	2	7	9	10	7	6	4	2	5	4	5	6	3	3	16	89

III.—Spirochæte-positive.

600	–	–	–	–	–	–	1	–	–	1	1	–	–	–	–	–	–	3
1,000	–	1	1	2	2	1	–	–	2	–	–	–	–	–	–	1	2	12
1,400	–	–	–	–	–	2	–	–	–	1	1	–	–	–	–	–	–	4
1,800	–	–	–	1	–	–	–	–	1	1	1	–	–	–	–	–	3	7
2,200	1	–	–	1	–	–	–	–	1	–	–	–	–	1	–	–	–	4
2,600	–	–	–	1	–	1	–	–	–	–	–	1	–	–	–	–	–	3
3,600	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	–	–	1
	1	1	1	5	2	5	1	–	4	3	3	1	–	1	–	1	5	34

VII.—(3) ANALYSIS OF TABLE V., AND LISTS OF CASES WITH EXCEPTIONALLY HEAVY OR EXCEPTIONALLY LIGHT ORGANS.

THE LIVER.

In the fresh non-spirochæte fœtuses :—

Total fœtuses	162
Total ratios	3,361
Mean ratio	21

In the macerated non-spirochæte fœtuses :—

Total fœtuses	95
Total ratios	3,347
Mean ratio	35

In this series of fœtuses, it can be shown, by taking the mean ratios in weight-groups of fresh fœtuses, that the age of the fœtus does not much influence the ratio.

Fresh fœtuses, 162 :—

600–1,800	Total ratios	564
	Total fœtuses	27
	Mean ratio	21
1,800-3,000	Total ratios	1,423
	Total fœtuses	67
	Mean ratio	21
3,000 and over	Total ratios	1,384
	Total fœtuses	68
	Mean ratio	20

The difference between the mean ratios in the fresh and macerated fœtuses is a striking one, and must be accounted for by the loss of weight of the liver by autolysis.

In all the spirochæte positive fœtuses, fresh and macerated :—

Total fœtuses	37
Total ratios	788
Mean ratio	21

In the macerated spirochæte positive fœtuses :—

Total fœtuses	35
Total ratios	755
Mean ratio	22

In the fresh spirochæte positive fœtuses :—

Total fœtuses	2
Total ratios	33
Mean ratio	16

*Examination of the exceptionally heavy and light livers of non-spirochæte fœtuses.**

The macerated series are of no use in considering the light livers, but are useful for the heavy ones.

In the fresh series, seven lie under the ratio of 15 :—

No. 42, R. 14, wt. of fœtus, 1,672, breech, cerebral hæmorrhage.
„ 143, R. 12, „ „ 2,000, accidental hæmorrhage, S +, T +.
„ 229, R. 13, „ „ 1,010, W +, osteochondritis, "certain syphilis."
„ 295, R. 12, „ „ 1,345, placenta prævia, S +, K +, T +.
„ 346, R. 14, „ „ 3,600, forceps.
„ 356, R. 14, „ „ 3,550, cerebral hæmorrhage.
„ 422, R. 14, „ „ 4,536, the liver contained a cyst, K —.

In the macerated series, three lie under the ratio of 15 :—

No. 152, R. 12, wt. of fœtus, 1,300, hydramnion, S +, K +; cause of death unknown.
„ 164, R. 6, „ „ 1,040, W +; cause of death unknown.
„ 234, R. 14, „ „ 2,200, W +, S +, K +, SR +; probable syphilis.

* The abbreviations used are the same as in Table X.

Additional heavy macerated :—

No. 105, R. 16, albuminuria of pregnancy, S +, SR +, K —, T —.
,, 190, R. 18, albuminuria of pregnancy, S +.
,, 247, R. 17, chronic renal disease, SR +.
,, 271, R. 19, placenta prævia.
,, 290, R. 17, probable syphilis, S +, K +, SR +, W +.
,, 309, R. 18, chronic renal disease.
,, 313, R. 19, certain syphilis, W +, S +.
,, 319, R. 17, cause of death unknown, K +, SR +.
,, 335, R. 19, accidental hæmorrhage, "probable" syphilis.
,, 370, R. 19, albuminuria of pregnancy.
,, 382, R. 17, albuminuria of pregnancy, K +, T +.

In the fresh series, five lie over the ratio of 30 :—

No. 13, R. 37, wt. of fœtus, 4,082, accidental hæmorrhage, SR —.
,, 159, R. 42, ,, ,, 1,710, accidental hæmorrhage, T +.
,, 194, R. 34, ,, ,, 2,600, prolapse of cord.
,, 209, R. 30, ,, ,, 3,400, cerebral hæmorrhage, SR —.
,, 225, R. 36, ,, ,, 1,800, extensive infarction of placenta.

SPLEEN RATIOS.

Fresh —Non-Spirochæte.

Weight of fœtus : —		
600–1,800	Total ratios	10,543
	Total fœtuses	27
	Mean ratio	390
1,800–3,000	Total ratios	22,357
	Total fœtuses	66
	Mean ratio	339
3,000 and over	Total ratios	24,713
	Total fœtuses	67
	Mean ratio	369
Total	Ratios	57,613
	Fœtuses	160
	Mean ratio	360

Macerated —Non-Spirochæte.

600–1,800	Total ratios	18,193
	Total fœtuses	34
	Mean ratio	533
1,800–3,000	Total ratios	19,631
	Total fœtuses	45
	Mean ratio	436
3,000 and over	Total ratios	6,910
	Total fœtuses	14
	Mean ratio	493
Total	Ratios	44,734
	Fœtuses	93
	Mean ratio	481

Spirochæte-Positive.

600–1,800	Total ratios	3,996
	Total fœtuses	22
	Mean ratio	182
1,800–3,000	Total ratios	2,260
	Total fœtuses	14
	Mean ratio	161
3,000 and over	Ratio	55
Total	Ratios	6,311
	Fœtuses	37
	Mean ratios	171

Unusually Light Spleens.

In the fresh fœtuses, five have a ratio of over 600 :—

No. 17, wt. of fœtus, 1,956, R. 652, placenta prævia.
„ 179, „ „ 4,500, R. 642, contracted pelvis.
„ 383, „ „ 1,160, R. 773, placenta prævia, SR +, K +.
„ 404, „ „ 2,820, R. 520, placenta prævia.
„ 421, „ „ 3,051 R. 612, cerebral hæmorrhage, SR —.

In the macerated fœtuses, thirteen have a ratio of over 700 :—

No. 30, wt. of fœtus, 4,149, R. 829, unknown, L —, K —, SR —.
„ 32, „ „ 3,032, R. 866, rt. hypogastric art. missing.
„ 150, „ „ 2,950, R. 1,475, unknown, K —.
„ 198, „ „ 1,640, R. 820, albuminuria of pregnancy, T —.
„ 217, „ „ 1,070, R. 713, albuminuria, retroplac. hæmatoma. SR —.
„ 228, „ „ 2,500, R. 714, retroplac. hæmatoma.
„ 257, „ „ 885, R. 885, chronic renal disease, SR —.
„ 270, „ „ 1,040, R. 693, unknown, L —.
„ 297, „ „ 759, R. 759, chronic renal disease, T —.
„ 321, „ „ 1,050, R. 700, unknown.
„ 323, „ „ 2,125, R. 350, unknown, small placenta, L —, K —.
„ 357, „ „ 2,067, R. 824, retroplacental hæmorrhage, L —.
„ 359, „ „ 2,105, R. 840, albuminuria of pregnancy, L —, T —.

Unusually Heavy Spleens.

In the fresh fœtuses, eleven have a ratio of under 200 :—

No. 87, wt. of fœtus, 3,340, R. 148, general œdema, Pl +, K —, SR —. T —.
„ 143, „ „ 2,000, R. 190, accidental hæmorrhage, L +, T +.
„ 145, „ „ 2,250, R. 187, mitral disease. Strangulation by cord.
„ 204, „ „ 2,590, R. 172, W +, accidental hæmorrhage, SR +.
„ 229, „ „ 1,010, R. 35, W +, "certain syphilis."
„ 245, „ „ 2,500, R. 192, transverse presentation.
„ 294, „ „ 2,170, R. 181, accidental hæmorrhage.
„ 295, „ „ 1,345, R. 192, placenta prævia, L +, K +, T +.
„ 350, „ „ 3,220, R. 178, contracted pelvis.
„ 355, „ „ 2,780, R. 173, cerebral hæm., SR +, T +.
„ 365, „ „ 1,100, R. 122, general œdema, SR +, T —.

In the macerated fœtuses, five have a ratio of under 200 :—

No. 105, wt. of fœtus, 1,510, R. 157, albuminuria of pregnancy, L +, SR +, T —, K —.
„ 152, „ „ 1,300, R. 185, hydramnion, L +, K +.
„ 190, „ „ 2,660, R. 133, albuminuria of pregnancy, L +.
„ 234, „ „ 2,200, R. 64, W +, L +, SR +, K + (probable syph.).
„ 247, „ „ 2,070, R. 165, albuminuria of pregnancy, L +, T +.

Five have a ratio of between 200 and 250 :—

No. 5, R. 207, hydrocephalus and spina bifida, K +, T —.
„ 34, R. 238, hypogastric artery absent, SR —.
„ 110, R. 212, albuminuria of pregnancy, K +.
„ 197, R. 210, accidental hæmorrhage, K —.
„ 290, R. 206, W +, L +, SR +, K + (probable syph.).

Eight have a ratio of between 250 and 300 :—

No. 65, R. 278, W +, "certain syphilis."
„ 68, R. 260, W +, unknown; possible syphilis.
„ 168, R. 283, unknown, SR −, T +.
„ 211, R. 263, W +, unknown; possible syphilis.
„ 313, R. 289, "certain syphilis," W +, L +.
„ 362, R. 290, albuminuria of pregnancy, SR +, K +.
„ 384, R. 265, unknown, SR +.
„ 403 (B), R. 294, albuminuria of pregnancy, SR +.

KIDNEYS.

Fresh—Non-Spirochæte.

600–1,800	Total ratios	3,421
	Total fœtuses	27
	Mean ratios	127
1,800–3,000	Total ratios	7,903
	Total fœtuses	68
	Mean ratio	115
3,000–End	Total ratios	8,815
	Total fœtuses	67
	Mean ratio	130
Total	Ratios	19,939
	Fœtuses	162
	Mean ratio	123

Macerated- Non-Spirochæte.

600–1,800	Total ratios	4,487
	Total fœtuses	32
	Mean ratio	140
1,800–3,000	Total ratios	6,379
	Total fœtuses	43
	Mean ratio	148
3,000–End	Total ratios	2,681
	Total fœtuses	16
	Mean ratio	168
Total	Ratios	13,547
	Fœtuses	91
	Mean ratio	149

Spirochæte-Positive.

600–1,800	Total ratios	2,316
	Total fœtuses	22
	Mean ratio	105
1,800–3,000	Total ratios	1,476
	Total fœtuses	14
	Mean ratio	105
3,000–End	Total ratios	103
	Total fœtuses	1
	Mean ratio	103
Total	Ratios	3,895
	Fœtuses	37
	Mean ratio	105

Exceptionally Heavy Kidneys.

Fresh fœtuses :—

No. 47, wt. of fœtus, 3,458, R. 89, accidental hæmorrhage.
„ 57, „ „ 2,400, R. 57, cerebral hæmorrhage.
„ 75, „ „ 2,820, R. 67, cause unknown, SR −, T +.
„ 177, „ „ 3,010, R. 72, breech labour.

No. 187, wt. of fœtus, 1,115, R. 47, placenta prævia, SR +, T +.
„ 226, „ „ 1,110, R. 69, breech, fœtal injuries.
„ 252, „ „ 2,050, R. 68, decapitation.
„ 259, „ „ 2,690, R. 68, cerebral hæmorrhage.
„ 295, „ „ 1,345, R. 64, placenta prævia, L +, T +.
„ 368, „ „ 2,460, R. 63, accidental hæmorrhage.
„ 374, „ „ 3,690, R. 84, placenta prævia, SR +.
„ 383, „ „ 1,160, R. 66, placenta prævia, S —, SR +.
„ 386, „ „ 3,120, R. 74, cerebral hæmorrhage.
„ 400, „ „ 3,010, R. 73, cerebral hæmorrhage.
„ 410, „ „ 3,150, R. 82, craniotomy.

Macerated fœtuses :—

No. 5, wt. of fœtus, 3,628, R. 107, hydrocephalus, S +, T —.
„ 110, „ „ 1,170, R. 82, albuminuria of pregnancy, S +.
„ 152, „ „ 1,300, R. 72, hydrammion, L +, S +.
„ 201, „ „ 2,400, R. 72, B. coli infection.
„ 234, „ „ 2,200, R. 98, probable syph., L +, S +, SR +.
„ 267, „ „ 2,700, R. 87, prolapsed cord.
„ 290, „ „ 2,585, R. 91, probable syph., L +, S +, SR +.
„ 319, „ „ 2,126, R. 72, albuminuria of pregnancy, L +, SR +.
„ 362, „ „ 3,770, R. 74, albuminuria of pregnancy, S +, SR +.
„ 382, „ „ 1,590, R. 82, albuminuria of pregnancy, L +, T +.

Exceptionally Light Kidneys.

Fresh fœtuses with ratio above 160 :—

No. 28, wt. of fœtus, 1,650, R. 162, eclampsia, SR —.
„ 87, „ „ 3,340, R. 219, general œdema, S +, SR —, T —.
„ 135, „ „ 4,300, R. 191, cerebral hæmorrhage, SR —.
„ 157, „ „ 2,990, R. 176, cerebral hæmorrhage.
„ 203, „ „ 3,350, R. 172, placenta prævia ; one kidney v. small.
„ 212, „ „ 2,100, R. 196, general œdema. SR —.
„ 232, „ „ 1,630, R. 776, kidneys remarkably small.
„ 248, „ „ 3,600, R. 195, craniotomy.
„ 288, „ „ 3,890, R. 181, prolapse of cord.
„ 302, „ „ 3,450, R, 192, placenta prævia, T +.
„ 303, „ „ 3,275, R. 173, placenta prævia.
„ 305, „ „ 2,580, R. 188, cerebral hæmorrhage, T —.
„ 315, „ „ 3,700, R. 224, prolapsed cord.
„ 337, „ „ 1,620, R. 162, cerebral hæmorrhage.
„ 395, „ „ 2,360, R. 169, general œdema, T —.
„ 422, „ „ 4,536, R. 181, cerebral hæmorrhage, L +.

Macerated fœtuses with ratio over 190 :—

No. 18, wt. of fœtus, 3,451, R. 192, cause unknown.
„ 30, „ „ 4,149, R. 207, cause unknown, L —, S —, SR —.
„ 49, „ „ 1,771, R. 208, eclampsia, SR —.
„ 60, „ „ 2,850, R. 208, one hypogastric and umbilical artery missing, SR —.
„ 105, „ „ 1,510, R. 215, albuminuria of pregnancy, L +, S +, SR +, T —.
„ 122, „ „ 2,050, R. 277, cause unknown ; suprarenal hæm.
„ 150, „ „ 2,950, R. 347, cause unknown, S —.
„ 170, „ „ 3,590, R. 211, placenta prævia, T +.
„ 197, „ „ 3,150, R. 210, accidental hæmorrhage, S +.

No. 238, wt. of fœtus, 4,880, R. 264, hydramnion, SR —.
„ 251, „ „ 1,750, R. 195, maternal pulmonary tubercle.
„ 273, „ „ 4,590, R. 195, cause unknown, SR —.
„ 278, „ „ 2,950, R. 190, small placenta, T +.
„ 292, „ „ 2,160, R. 309, probable syphilis.
„ 323, „ „ 2,125, R. 304, cause unknown, L —, S —.
„ 336, „ „ 1,010, R. 202, cause unknown.
„ 388, „ „ 2,110, R. 242, cause unknown.
„ 412, „ „ 3,120, R. 240, hypogastric and umbilical art. absent.

SUPRARENALS.

Fresh—Spirochæte-free.

600–1,800	Total ratios	9,352
	Total fœtuses	27
	Mean ratio	346
1,800–3,000	Total ratios	27,972
	Total fœtuses	67
	Mean ratio	417
3,000–End	Total ratios	27,002
	Total fœtuses	66
	Mean ratio	410
Total	Ratios	64,326
	Cases	160
	Mean ratio	402

Macerated—Spirochæte-free.

600–1,800	Total ratios	13,287
	Total fœtuses	31
	Mean ratio	429
1,800–3,000	Total ratios	21,517
	Total fœtuses	43
	Mean ratio	500
3,000–End	Total ratios	8,606
	Total fœtuses	16
	Mean ratio	538
Total	Ratios	43,410
	Fœtuses	90
	Mean ratio	482

Spirochæte-Positive.

600–1,800	Total ratios	7,243
	Total fœtuses	21
	Mean ratio	345
1,800–3,000	Total ratios	4,981
	Total fœtuses	13
	Mean ratio	383
3,000–End	Total ratios	509
	Total fœtuses	1
	Mean ratio	509
Total	Ratios	12,733
	Fœtuses	35
	Mean ratio	364

Exceptionally Heavy Suprarenals.

Amongst the fresh fœtuses, there are fourteen examples of exceptionally heavy suprarenal :—

No. 103, wt. of fœtus, 2,450, R. 181, placenta prævia.
„ 111, „ „ 3,320, R. 246, cerebral hæmorrhage.
„ 160, „ „ 3,400, R. 262, premature rupture of membranes.
„ 169, „ „ 3,590, R. 244, prolapsed cord.
„ 187, „ „ 1,115, R. 171, placenta prævia, K +, T +.
„ 204, „ „ 2,590, R. 235, W +, accidental hæmorrhage, S +.
„ 268, „ „ 2,265, R. 216, cerebral hæmorrhage.
„ 339, „ „ 4,380, R. 213, cerebral hæmorrhage, T +.
„ 355, „ „ 2,780, R. 206, cerebral hæmorrhage, S +, T +.
„ 365, „ „ 1,100, R. 177, placenta prævia, S +, T —.
„ 373, „ „ 1,870, R. 215, placenta prævia.
„ 374, „ „ 3,690, R. 209, placenta prævia, K +.
„ 383, „ „ 1,160, R. 178, placenta prævia, S —.
„ 404 (B), „ „ 2,820, R. 165, placenta prævia.

Amongst the macerated fœtuses, there are ten examples :—

No. 105, wt. of fœtus, 1,510, R. 244, albuminuria of pregnancy, L +, S +, K —, T —.
„ 189, „ „ 1,180, R. 236, cause unknown.
„ 234, „ „ 2,200, R. 232, probable syphilis, L +, S +, K +.
„ 290, „ „ 2,585, R. 253, probable syphilis, L +, S +, K +.
„ 319, „ „ 2,126, R. 250, cause unknown, L +, K +.
„ 362, „ „ 3,770, R. 191, albuminuria of pregnancy, L +, K +.
„ 384, „ „ 2,650, R. 212, cause unknown, S +.
„ 394, „ „ 1,138, R. 228, cause unknown, L —.
„ 403 (B), „ „ 2,500, R. 238, albuminuria of pregnancy, S +.
„ 407, „ „ 2,480, R. 248, transverse presentation.

Exceptionally Light Suprarenals.

Fourteen fresh fœtuses have exceptionally light suprarenals :—

No. 13, wt. of fœtus, 4,082, R. 628, accidental hæmorrhage, L —.
„ 28, „ „ 1,650, R. 1,100, eclampsia, K —.
„ 75, „ „ 2,820, R. 940, fœtal ascites, K +, T +.
„ 82, „ „ 3,000, R. 600, placenta prævia.
„ 87, „ „ 3,340, R. 607, general œdema of fœtus, S +, K —, T —.
„ 107, „ „ 3,150, R. 788, cause unknown.
„ 116, „ „ 2,050, R. 602, placenta prævia, T +.
„ 135, „ „ 4,300, R. 782, cerebral hæmorrhage, K —.
„ 137, „ „ 2,900, R. 645, B. coli infection.
„ 188, „ „ 2,200, R. 1,570, anencephaly, T +.
„ 209, „ „ 3,400, R. 755, cerebral hæmorrhage, L —.
„ 212, „ „ 2,100, R. 1,400, general œdema of fœtus, K —.
„ 301, „ „ 3,540, R. 644, cerebral hæmorrhage.
„ 421, „ „ 3,061, R. 612, cerebral hæmorrhage, S —.

Twelve macerated fœtuses have exceptionally light suprarenals :—

No. 30, wt. of fœtus, 4,149, R. 1,037, small placenta, L —, S —, K —.
„ 34, „ „ 2,621, R. 874, small placenta, S +.
„ 49, „ „ 1,771, R. 737, eclampsia, K —.
„ 60. „ „ 2,850, R. 712, absence of one hypogastric and umbilical artery, K —.
„ 122, „ „ 2,050, R. 820, hæmorrhage into left suprarenal capsule, L —, K —.

No. 168, wt. of fœtus, 2,550, R. 945, B. coli. infection, S +, T +.
,, 215, ,, ,, 2,580, R. 860, cause unknown.
,, 221, ,, ,, 2,200, R. 880, cause unknown.
,, 230, ,, ,, 4,050, R. 900, postmaturity, L —.
,, 238, ,, ,, 4,880, R. 751, cause unknown, K —.
,, 257, ,, ,, 885, R. 885, chronic renal disease.
,, 273. ,, ,, 4,590, R. 706, small placenta, K —.

THYMUS.

Fresh—Spirochæte-free.

Fœtal weight in grms. :—

600–1,800	Total ratios	10,718
	Total fœtuses	26
	Mean ratio	412
1,800–3,000	Total ratios	25,962
	Total fœtuses	67
	Mean ratio	387
3,000–End	Total ratios	25,745
	Total fœtuses	66
	Mean ratio	390
Total	Ratios	62,425
	Fœtuses	159
	Mean ratio	393

Macerated—Spirochæte-free.

600–1,800	Total ratios	23,454
	Total fœtuses	30
	Mean ratio	782
1,800–3,000	Total ratios	24,126
	Total fœtuses	43
	Mean ratio	561
3,000–End	Total ratios	10,085
	Total fœtuses	16
	Mean ratio	630
Total	Ratios	57,665
	Fœtuses	89
	Mean ratio	648

Spirochæte-Positive.

600–1,800	Total ratios	9,575
	Total fœtuses	19
	Mean ratio	504
1,800–3,000	Total ratios	7,858
	Total fœtuses	14
	Mean ratio	561
3,000–End	Total ratios	440
	Total fœtuses	1
	Mean ratio	440
Total	Ratios	17,873
	Fœtuses	34
	Mean ratio	526

Exceptionally Heavy Thymus.

Fresh fœtuses, with ratio of under 200 :—

No. 51, R 162, cerebral hæmorrhage.
,, 75, R 156, cause unknown, K +, SR —.
,, 116, R 178, placenta prævia, SR —.
,, 136, R 175, placenta prævia.
,, 143, R 117, accidental hæmorrhage, L +, S +.
,, 159, R 171, accidental hæmorrhage, L —.
,, 187, R 101, placenta prævia, K +, SR +.
,, 188, R 142, anencephaly, SR —.
,, 206, R 176, anencephaly.
,, 265, R 151, placenta prævia.
,, 295, R 96, placenta prævia, L +, S +, K +.
,, 302, R 181, placenta prævia, K —.
,, 339, R 183, cerebral hæmorrhage, SR +.
,, 340, R 185, cerebral hæmorrhage.
,, 343, R 144, placenta prævia.
,, 344, R 173, placenta prævia.
,, 349, R 172, craniotomy.
,, 355, R 146, cerebral hæmorrhage, S +, SR +.
,, 406, R 167, cerebral hæmorrhage.

Macerated fœtuses, with ratio of under 300 :—

No. 168, R 255, B. coli infection, S +, SR —.
,, 170, R 231, placenta prævia, K —.
,, 247, R 295, chronic renal disease, L +, S +.
,, 278, R 295, small placenta, K —.
,, 281, R 280, maternal pneumonia.
,, 282, R 281, accidental hæmorrhage.
,, 308, R 257, transverse presentation.
,, 367, R 222, accidental hæmorrhage.
,, 382, R 289, albuminuria of pregnancy, L +, K +.

Exceptionally Light Thymus.

Fresh fœtuses, with ratios of over 800 :—

No. 35, wt. of fœtus, 3,968, R 1,133, double cystic fœtal kidneys.
,, 48, ,, ,, 1,300, R 812, eclampsia.
,, 87, ,, ,, 3,340, R 2,226, general œdema of fœtus, S +, SR —, K —.
,, 298, ,, ,, 2,894, R 826, cause unknown.
,, 305, ,, ,, 2,580, R 860, cerebral hæmorrhage, K —.
,, 334, ,, ,, 2,058, R 1,210, cause unknown.
,, 365, ,, ,, 1,100, R 2,200, general fœtal œdema, S +, SR +.
,, 395, ,, ,, 2,360, R 3,371, general fœtal œdema, K —.
,, 419, ,, ,, 3,430, R 857, postmaturity, K +.

Macerated fœtuses, with ratios of over 1,200 :—

No. 5, wt. of fœtus, 3,628, R 1,577, hydrocephalus, spina bifida, S +, K +.
,, 38, ,, ,, 2,494, R 1,467, albuminuria of pregnancy.
,, 105, ,, ,, 1,510, R 1,680, albuminuria of pregnancy, L +, S +, SR +, K —.
,, 198, ,, ,, 1,640, R 1,640, albuminuria of pregnancy, S —.
,, 262, ,, ,, 1,800, R 1,200, accidental hæmorrhage.
,, 280, ,, ,, 780, R 1,560, placental hæmorrhage, accidental hæmorrhage.
,, 297, ,, ,, 759, R 1,518, chronic renal disease, S —.
,, 359, ,, ,, 2,100, R 1,400, albuminuria of pregnancy, L —, S —.

VII. (4) The Length-Weight-Ratio of the Fœtus.

This ratio is got by dividing the weight of the fœtus by its length, and indicates its state of nutrition. The ratio is high if the fœtus is heavy, and low if it is light in proportion to its length.

The ratios for fresh, macerated and syphilitic fœtuses respectively, have been arranged in groups differing by 5 in Table VI. to show their frequency-distribution amongst fœtuses of different weight-groups. The mean ratios have also been calculated, and are shown in Table VI.

The tables show that the ratio increases progressively with the length of the fœtus; a premature fœtus is relatively light, and a mature fœtus relatively heavy in proportion to its length. It is also apparent that the mean ratios for fresh and macerated fœtuses show very little difference.

It was especially desirable to find out if syphilis had a bad effect on the state of fœtal nutrition. The tables show that the mean ratios for syphilitic fœtuses coincide very closely with those for non-syphilitic, and that an exceptionally light fœtus is no more likely to be syphilitic than non-syphilitic.

Those fœtuses whose ratios show extreme deviations from the mean may now be selected, in order to find out if there is a noteworthy association between exceptional fœtal heaviness or lightness and pathological conditions of the mother, fœtus or placenta. Details will readily be found by reference to the tables.

1. *Exceptionally Heavy Fœtuses.*—21 fresh fœtuses (Nos. 103, 135, 156, 163, 179, 188, 220, 235, 288, 314, 315, 326, 331, 339, 340, 341, 343, 344, 374, 395, 422) and 12 macerated (Nos. 18, 150, 190, 211, 230, 238, 247, 273, 278, 313, 359, 371) show exceptional heaviness. Examination of these cases is disappointing in that no generalizations can be drawn. Amongst the fresh fœtuses are five cases of placenta prævia, one of fœtal general œdema, one of anencephaly, three of contracted pelvis: the obstetrical complications include five cases of placenta prævia, three of contracted pelvis, and others of no particular significance; five of the fœtuses have exceptionally light kidneys, three have heavy thymus glands, and three heavy suprarenals.

Amongst the macerated fœtuses, in four the cause of death is unknown, two are cases of probable syphilis, two of pregnancy-kidney and chronic renal disease; amongst the organ-weights, there is again a preponderance of light kidneys, five fœtuses showing that peculiarity.

2. *Exceptionally Light Fœtuses.*—Eleven fresh fœtuses (Nos. 48, 57, 140, 245, 263, 272, 283, 286, 295, 406, 409) and 13 macerated (Nos. 11, 44, 68, 217, 224, 257, 262, 270 271, 280, 297, 376, 392) show exceptional lightness.

Again, it is disappointing that no generalizations can be made after studying the cases. Amongst the fresh fœtuses are

two cases of eclampsia, two of albuminuria, two of placenta prævia and other cases of the usual obstetrical complications; the fœtal organ weights reveal nothing of interest. Amongst the macerated fœtuses nine have albuminuric states, comprising two cases of chronic renal disease, one of albuminuria, and three of accidental hæmorrhage; amongst the organ weights are three exceptionally light thymus glands.

Thus, the search for any correlation between exceptional fœtal weight and other abnormalities is disappointing. The only notable point that emerges is that 10 out of 35 heavy fœtuses have exceptionally light kidneys.

VII.—(5) TABLE VI.

Tables showing Frequency-Distribution of the Length-weight Ratios amongst Fœtuses of different Weights.

Fresh.

Weight of Fœtus.	Ratios.											Number of Fœtuses.
	20	35	40	45	50	55	60	65	70	75	80	
600 -	—	—	—	—	—	—	—	—	—	—	—	—
1,000 -	15	—	—	—	—	—	—	—	—	—	—	15
1,400 -	—	4	5	1	—	—	—	—	—	—	—	10
1,800 -	—	1	6	13	1	1	—	—	—	—	—	22
2,200 -	—	—	—	8	8	2	2	—	—	—	—	20
2,600 -	—	—	—	—	8	13	2	1	—	—	—	24
3,000 -	—	—	—	—	—	12	14	10	1	—	—	37
3,400 -	—	—	—	—	—	—	3	9	4	2	—	18
3,800 -	—	—	—	—	—	—	1	—	2	1	2	6
4,200 -	—	—	—	—	—	—	—	—	—	—	6	6
	15	5	11	22	17	28	22	20	7	3	8	158

Macerated.

600 -	6	—	—	—	—	—	—	—	—	—	—	6
1,000 -	16	2	—	—	—	—	—	—	—	—	—	18
1,400 -	—	5	3	1	—	—	—	—	—	—	—	9
1,800 -	—	3	1	7	4	1	—	—	—	—	—	16
2,200 -	—	—	—	8	7	3	—	—	—	—	—	18
2,600 -	—	—	—	—	1	4	3	2	—	—	—	10
3,000 -	—	—	—	—	—	—	4	—	—	—	—	4
3,400 -	—	—	—	—	—	—	—	3	1	1	—	5
3,800 -	—	—	—	—	—	—	—	—	1	1	—	2
4,200 -	—	—	—	—	—	—	—	—	—	—	2	2
	22	10	4	16	12	8	7	5	2	2	2	90

Syphilis.

Weight of Fœtus.	Ratios.											Number of Fœtuses.
	30	35	40	45	50	55	60	65	70	75	80	
600 -	4	—	—	—	—	—	—	—	—	—	—	4
1,000 -	11	3	—	—	—	—	—	—	—	—	—	14
1,400 -	2	1	1	—	—	—	—	—	—	—	—	4
1,800 -	—	—	1	4	2	—	—	—	—	—	—	7
2,200 -	—	—	—	—	1	4	—	—	—	—	—	5
2,600 -	—	—	—	—	—	2	1	—	—	—	—	3
3,000 -	—	—	—	—	—	—	—	1	—	—	—	1
	17	4	2	4	3	6	1	1	—	—	—	38

MEAN LENGTH-WEIGHT RATIOS.

Fœtal Weight.	*Fresh.*		*Macerated.*		*Syphilis.*	
	No.	Mean R.	No.	Mean R.	No.	Mean R.
600-1,000 -	—	—	6	30	4	20
1,000–1,400 -	15	30	18	30	14	30
1,400–1,800 -	10	40	9	39	4	37
1,800–2,200 -	22	47	16	46	7	50
2,200–2,600 -	20	52	18	51	5	55
2,600–3,000 -	24	57	10	60	3	58
3,000–3,400 -	37	63	4	62	1	66
3,400–3,800 -	18	69	5	71	—	—
3,800–4,200 -	6	75	2	75	—	—
over 4,200 -	6	84	2	89	—	—

SECTION VIII.

THE PLACENTA.

(1) The Weight-ratios of the Placenta. (Tables VII., VIII., IX., X.)
(2) Relative Placental Insufficiency.
(3) Abnormal Placental States as Causes of Fœtal Death.
(4) The Syphilitic Placenta. (Table XI.)

VIII.—(1) WEIGHT-RATIOS OF THE PLACENTA.

The placentas were weighed after the membranes and cord had been removed. The weight-ratio is given by dividing the weight of the fœtus by the weight of the placenta. The mean

ratios for different groups of fœtuses have been calculated and arranged in Tables VII., VIII. and IX.

Table VII. shows the mean ratios for fresh and macerated non-syphilitic fœtuses respectively, (*a*) the mean for all fœtuses, (*b*) the mean when the fœtuses are divided into three weight-groups.

The mean for fresh and macerated fœtuses is seen to be much the same. The weight grouping shows that the ratio becomes gradually higher as the weight of the fœtus increases; that is to say, the placenta is relatively heavy in premature fœtuses. This is an important point to remember when considering the heaviness of the syphilitic placenta, for syphilitic fœtuses are nearly all premature.

Table VIII. shows the mean ratios for Syphilitic Placentas: (*a*) the mean for all; (*b*) the mean when the fœtuses are divided into weight-groups.

Table IX. shows, grouped together, the ratios for fresh and macerated non-syphilitic, and for syphilitic fœtuses.

Table X. shows the ratios arranged so as to show their frequency-distribution amongst fœtuses of different weights. This provides a ready means for picking out the extremes at either end of the scale (*i.e.*, the exceptionally heavy and exceptionally light placentas). The great irregularity in the distribution of the ratios shows what a variable weight the placenta has even in fœtuses of approximately the same weight. This table will be of particular use when the non-syphilitic are compared with the syphilitic placentas. A point brought out is that there is a relatively large number of exceptionally light placentas amongst the macerated fœtuses; with this exception, there is little difference between the ratios in the two groups.

TABLE VII.—WEIGHT-RATIOS OF THE PLACENTA: NON-SYPHILITIC.

(*a*)

Fresh non-syphilitic.		*Macerated non-syphilitic.*	
Number of fœtuses	94	Number of fœtuses	64
Mean ratio	8(7·6)	Mean ratio	8(7·9)

(*b*)

Fresh.

	Under 1,800 grm.	1,800 to 3,000 grm.	Over 3,000 grm.
Number of fœtuses	20	36	38
Mean ratio	6	8	8
Macerated.			
Number of fœtuses	18	33	13
Mean ratio	6	8	10

TABLE VIII.—WEIGHT-RATIOS OF THE PLACENTA: SYPHILITIC.

(*a*)

Number of fœtuses	34
Mean ratio	6(5·6)

(*b*)

	Under 1,800 grm.	1,800 to 3,000 grm.
Number of fœtuses	19	15
Mean ratio	5(4·6)	7(6·8)

TABLE IX.—WEIGHT-RATIOS OF THE PLACENTA: ALL TOGETHER.

	Number.	Mean ratio for all.	Under 1,800 grm.	1,800 to 3,000 grm.	Over 3,000 grm.
Fresh non-syphilitic	94	8	6	8	8
Macerated non-syphilitic	64	8	6	8	10
Syphilitic	34	6	5	7	—

TABLE X.—RATIOS ARRANGED IN TABLES SHOWING FREQUENCY-DISTRIBUTION AMONGST FŒTUSES OF DIFFERENT WEIGHTS.

FRESH NON-SYPHILITIC.

Weight of Fœtus.	Ratio.											Total.
	2	3	4	5	6	7	8	9	10	11	12	
600 to 1,800	2	0	2	8	3	2	2	0	1	0	0	20
1,800 to 3,000	0	0	0	2	5	7	6	11	4	1	0	36
Over 3,000	0	0	0	1	4	11	6	9	2	4	1	38
	2	0	2	11	12	20	14	20	7	5	1	94

MACERATED NON-SYPHILITIC.

600 to 1,800	0	1	5	2	1	4	1	2	2	0	0	18
1,800 to 3,000	0	1	2	4	2	7	4	8	0	2	3	33
Over 3,000	0	0	0	0	0	1	2	4	0	3	3	13
	0	2	7	6	3	12	7	14	2	5	6	64

SYPHILITIC.

600 to 1,800	0	7	3	3	3	3	0	0	0	0	0	19
1,800 to 3,000	0	2	1	4	1	1	3	0	2	1	0	15
	0	9	4	7	4	4	3	0	2	1	0	34

I. *Unusually Heavy Placentas, excluding Syphilis.*

For this purpose I have selected placentas with a weight-ratio of under 3·6 in fœtuses of less than 1,800 grms. weight, with a ratio of under 5·6 for fœtuses from 1,800 to 3,000 grms., and with a ratio of under 6·5 for fœtuses of over 3,000 grms.

600 *to* 1,800 *grm. :* 3 *Placentas.*

(a) *Fresh.*

No. 48 : Ratio 1·8.—Case of eclampsia. The placenta contained interstitial hæmorrhages and showed much red infarction.

No. 365 : Ratio 1·8.—Case of general œdema of fœtus. There had been many former fœtal deaths. This time there was accidental hæmorrhage. Microscopical section shows the villi enlarged, avascular and with dense stroma; resembling the syphilitic placenta, but the stroma is denser and the villi more irregular.

(b) *Macerated.*

No. 291 : Ratio 3·3.—A case of accidental hæmorrhage. The placenta contained an interstitial hæmorrhage and red infarction.

1,800 *to* 3,000 *grm. :* 8 *Placentas.*

(a) *Fresh.*

No. 360 : Ratio, 5·2.—Case of antepartum hæmorrhage, probably placenta prævia.

(b) *Macerated.*

No. 68 : Ratio, 3·2, W +, Possible syphilis.

No. 190 : Ratio, 3·8.—Case of albuminuria of pregnancy; placenta weighed 700 grms. and was the largest in the series. Contained two interstitial hæmorrhages. The villi were enlarged, with very loose stroma.

No. 335 : Ratio, 4·3.—Case of accidental hæmorrhage. The villi are large and resemble exactly those typical of syphilis. "Probable Syphilis."

No. 168 : Ratio, 5·5.—Clot, probably ante mortem, in umbilical vein, coliform bacilli grown from fœtal blood. Villi normal.

No. 282 : Ratio, 5·5.—Case of accidental hæmorrhage. Much white infarction and some red.

No. 290 : Ratio, 5.—Probably syphilis. W +. Spleen and liver enlarged; otherwise no signs of syphilis. Villi normal.

No. 292 : Ratio, 5·4.—Cause of death unknown. The patient had formerly had syphilis. Villi normal.

3,000 *grm. and over :* 4 *Placentas.*

(a) *Fresh.*

No. 87 : Ratio, 5·5.—General œdema of fœtus. Villi large and irregular; stroma dense and with round-celled infiltration.

No. 108: Ratio, 5·9.—Albuminuria of pregnancy. Villi normal.

No. 311: Ratio, 6·3.—Case of forceps delivery, death of fœtus from cerebral hæmorrhage. The placental decidua showed gelatinous degeneration.

No. 401: Ratio, 5·8.—Breech-labour. Fœtal death from cerebral hæmorrhage. Placenta showed red infarction otherwise was normal.

Summary of Unusually Heavy Placentas.

1. Two cases of probable (No. 290, 335) and one of possible syphilis (No. 68), both with positive maternal Wassermann reaction, but no spirochætes and no osteochondritis in fœtus. One case (No. 292) with a history of maternal syphilis, but no signs in mother or fœtus.

2. Six cases of toxæmia of pregnancy, comprising two of albuminuria (Nos. 108, 190), one of eclampsia (No. 48) and three of accidental hæmorrhage (Nos. 282, 291, 335). In most of these the placenta showed red infarcts and interstitial hæmorrhages.

3. Two cases of general œdema of the fœtus (Nos. 87, 365).

4. One case of placenta prævia.

5. Two cases of difficult labour resulting in fœtal cerebral hæmorrhage, and one case in which the cause of death was unknown.

II. *Unusually Light Placentas, excluding Syphilis.*

For this purpose I have selected the 17 placentas with a weight ratio of over 10·6.

1,800 *to* 3,000 *grm.*

(a) *Fresh.*

No. 373: Ratio, 11 —Case of placenta prævia.

(b) *Macerated.*

No. 34: Ratio, 11 —Cause of death unknown. Villi normal.

No. 278: Ratio, 11·8.—Cause of death unknown.

No. 322: Ratio, 10·7.—Cause of death unknown.

No. 323: Ratio, 12·5.—Cause of death unknown.

No. 387: Ratio, 12·3.—Cause of death unknown.

No. 185: Ratio, 11.—W +. "Possible" syphilis.

3,000 *grm. and over.*

(a) *Fresh.*

No. 82: Ratio, 11·5.—Case of placenta prævia.

No. 191: Ratio, 12.—Case of accidental hæmorrhage.

No. 236: Ratio, 11·2.—Case of placenta prævia.

No. 331: Ratio, 10·8.—Case of obstructed labour.

(b) *Macerated.*

No. 30 : Ratio, 12·9.—Cause of death unknown.
No. 32 : Ratio, 11·2.—Right hypogastric artery missing. Villi normal.
No. 180 : Ratio, 10·8.—Case of placenta prævia
No. 184 : Ratio, 11.—Cause of death unknown.
No. 371 : Ratio, 13·3.—Cause of death unknown.
No. 273 : Ratio, 13·5.—Cause of death unknown.

Summary of unusually Light Placentas.

1. The very noteworthy fact emerges that 10 of these placentas belonged to macerated fœtuses, for which no other cause of death can be found; if, indeed, the smallness of the placenta is not taken as the cause of death, these fœtuses must be classed under "cause of death unknown." This subject will be discussed more fully below.

2. In one fœtus the hypogastric and umbilical artery of one side was missing.

3. The others call for no particular comment: four cases of placenta prævia and one of accidental hæmorrhage.

VIII.—(2) Relative Placental Insufficiency.

The placenta is an organ peculiarly liable to degenerations and other vicissitudes. Were it not for the provision of a generous margin of safety by the placental tissue being in excess of the actual requirements of the fœtus, the fœtus would have a precarious existence. A remarkable example of this, in the present series, is case No. 225, where, although at least two-thirds of the placental tissue had been destroyed by white necrosis ("white infarction"), the fœtus was able to maintain its existence until labour was almost completed.

A consideration of two elementary points seems to show that the oxygen-requirement of the fœtus must be small compared to that of postnatal existence; (1) in postnatal life, at each cardiac systole, the volume of blood passing through the lungs by the pulmonary artery is equal to the volume passing into the systemic circulation by the aorta; whereas in the fœtus, the volume of blood passing through the placenta by the two small hypogastric arteries is only a small fraction of the systemic blood; (2) the oxygen content of the alveolar air is high compared to that of the maternal blood. I am aware that these considerations cannot be accepted as proof of the low oxygen requirement of the fœtus until something definite is known of the relative rate of absorption of oxygen, from the maternal blood by the placenta on the one hand, and from the alveolar air by the lungs on the other.

Another fact, well known to all who have practised scopolamine-morphine narcosis, is the length of time a fœtus, after birth and separation, can remain in a state of apnœa; I have

personally seen fœtuses maintain their fœtal existence (*i.e.* no pulmonary respiration) for twenty minutes, living on their store of reserve oxygen, and then begin to breathe and cry with vigour, having suffered nothing.

The fœtus requires a minimum amount of healthy placental tissue to maintain life; in cases where this amount is not available we speak of "relative placental insufficiency."

Very obvious examples of fœtal death from relative placental insufficiency are separation of the placenta in cases of placenta prævia and accidental hæmorrhage.

The intrinsic morbid states of the placenta which may give rise to placental insufficiency are the following :—

(1) Extensive red infarction. (2) Extensive white infarction. (3) Intraplacental hæmorrhage. (4) Retroplacental hæmatoma. (5) Avascularity of the villi as found in syphilis. (6) An excess of the normal degenerative changes, as sometimes found in the placentas of postmature fœtuses. (7) An abnormally small placenta. (8) The diminished fœto-placental circulation caused by absence of one hypogastric and umbilical artery.

These several states, and their liability to cause relative placental insufficiency, will be described under their proper headings.

There are two further considerations. If a large part of the placental tissue is destroyed, the reserve power of the placenta may be dangerously lowered; the margin of safety left for further events, such as retroplacental hæmatoma or an excess of normal degeneration, may be inadequate. Secondly, the amount of healthy placental tissue may be enough for the requirements of the fœtus during the quiescent period of pregnancy, but not sufficient for the greater demands of labour, especially a long one. Case No. 225 is an example. During uterine contractions the uteroplacental circulation is impeded; the fœtal heart beat, also, is slowed and the oxygen carrying power of the blood thereby retarded.

VIII.—(3) On Abnormal Placental States as Causes of Fœtal Death.

No post mortem on a fœtus is complete without the examination of its most important organ. It would be strange, indeed, if the investigation of cases of fœtal death did not commonly reveal abnormalities, both of structure and of attachment, in an organ so vital to fœtal existence, and so liable to degenerative changes both of its fœtal and maternal elements. Separation of the placenta, in cases of placenta prævia and accidental hæmorrhage, are not included in the terms of this section; but it may be mentioned that 35 cases of placenta prævia and 31 of accidental hæmorrhage occurred amongst the 300 cases.

The changes which may be found by examining the placenta, in the light of present knowledge, as direct causes of fœtal death or as evidence of other causes, are as follows :—

1. Retroplacental hæmorrhage, or hæmatoma, as evidenced by clots of various age, or by the depressions on the maternal surface caused by them.
2. Intraplacental hæmorrhages.
3. Extensive white infarction.
4. Extensive red infarction—found almost always in association either with retroplacental hæmorrhage or toxæmia of pregnancy, or both.
5. Syphilitic changes in the villi.
6. An unusually small placenta.
7. Absence of one artery from the umbilical cord associated with absence of the corresponding hypogastric artery of the fœtus.

Some of these changes are sufficient to kill the fœtus by their direct action, *i.e.* retroplacental hæmatoma; others, such as syphilitic changes in the villi or red infarction, are indicative of other lethal conditions.

An immense amount of work still requires to be done on the placenta, histological, chemical and bacteriological, and later knowledge is sure to reveal other morbid placental states which either act themselves as direct causes of fœtal death, or supply evidence of other causes.

In the light of present knowledge, the above is a fairly representative list; at all events, it comprises all the points considered in the present investigation.

Histology of the Placenta.

The points especially to be noticed, in microscopical sections, are: (1) the size of the terminal villi and their spacing, *i.e.* whether they show the normal pattern or are closely crowded; (2) the bloodvessels of the terminal villi; (3) the stroma—its density and degenerations; (4) the bloodvessels of the larger chorionic stems; (5) infarction, white or red; (6) the chorionic epithelium—hypertrophy and budding. I have experienced great difficulty in interpreting many placental sections, and know of no comprehensive account of placental histology; it is worthy of a monograph with a wealth of illustrations.

(1) *The Size of the Terminal Villi.*—In the healthy placenta most of the terminal stems are of fairly uniform size and are, in this respect, in contrast to the larger chorionic stems. Great increase in size of the terminal villi is found in syphilis and general œdema of the fœtus; many cases of pregnancy-toxæmia show the same change in lesser degree.

(2) *The Blood vessels of the Terminal Villi.*—These are fine endothelial-walled tubes lying free in the stroma. They are numerous and, moderately distended with blood, fill about

half the area of the villus. When collapsed they are still discernible as small circular aggregations of cells. I have never yet seen any evidence of endarteritis in the bloodvessel of a terminal villus; nor can I conceive of such a process, since the tissue-elements necessary for it are wanting. In syphilis, the vessels of the terminal villi are conspicuous by their absence; in early red infarction they are characteristically engorged with blood.

(3) *The Stroma of the Villus* consists of a groundwork of collagen fibres containing scanty round and ovoid cells. The stroma is normally loose. In syphilis the stroma is dense and the cells more numerous. A characteristic hyaline change appears in the stroma of some of the terminal villi towards the end of pregnancy. This, in moderation, is a normal state of the ripe placenta. In the placentas of macerated fœtuses this change is so intense as to be almost universal, affecting almost every villus, and is so evident as itself to betray that the placenta showing it has belonged to a fœtus that has been retained in the uterus for a considerable period after death. This hyaline degeneration is no doubt a necrosis; the nuclei of the stroma cells disappear and the collagen fibres swell into a uniform glassy matrix which has a strong affinity for eosine.

(4) *The Bloodvessels of the Larger Chorionic Stems.*—These undergo obliterative changes during the last two or three months of pregnancy. No reliance can be placed on an excess of this process as a diagnostic point. It may be found equally developed in the placentas of fœtuses dead from a diversity of causes, *i.e.* in cases of fœtal death due to traumatism of labour, syphilis, pregnancy-toxæmia and other causes. It is probable that no special significance belongs to an excess of this process as a diagnostic point. But these vascular changes are worth a special research.

(5) *The Chorionic Epithelium.*—In the placentas of macerated fœtuses which have been long retained after death the chorionic epithelium is often found proliferated in the form of little densely nucleated buds or masses which stain a very dark blue colour with hæmatoxylin; the masses usually have a densely granular appearance. The epithelium of contiguous villi frequently appears to coalesce, and the villi, in places, appear to be bound together by granular darkly-staining material.

The placentas of most macerated fœtuses are characterized by three changes in the villi: excessive hyaline degeneration of the stroma; collapse of the blood-vessels and, ultimately, disappearance of the endothelium from hyaline degeneration; and overgrowth of the chorionic epithelium in the form of granular and densely nucleated masses. It is important to recognize these changes as consequences and not as causes of fœtal death.

The abnormal placental states found amongst the 192 placentas examined will now be severally considered.

Red Infarction.

There were 31 well recognizable examples of red infarction; 12 in the placentas of fresh fœtuses (Nos. 13, 28, 48, 160, 167, 191, 277, 286, 293, 338, 379, 401) and 19 in those of the macerated fœtuses (Nos. 7, 11, 26, 44, 110, 127, 133, 192, 197, 217, 228, 280, 282, 287, 291, 335, 357, 412, 416).

Red infarcts are areas of localized placental death. They represent the early stage of this process; later on, if time allows the development of successive stages, they gradually become lighter in colour and firmer in texture, and ultimately are converted into the better known and commoner white infarcts. Red infarcts are recognizable as circumscribed areas, darker in colour and firmer in texture than the surrounding paler and more spongy normal placental tissue (Fig. 16). Very often they are difficult to recognize in the fresh placenta, but stand out clearly after the placenta has been washed in running water for a short time and has lain in formalin solution for a week or more; this treatment allows the blood in the intervillous spaces to become diffused out. With the escape of blood the cut surface of the healthy placenta becomes paler and the infarct-areas remain dark and stand out clearly against the paler background. Under the microscope the red areas show characteristic appearances. The bloodvessels of the villi are enormously distended with blood and the villi are closely packed together; between the villi there is often a deposit of fibrin.

Red infarcts of the placenta vary in number and size; they are usually ovoid or wedge-shaped areas, extending through part or the whole thickness of the placenta, and in the great majority of cases their base lies against the decidual surface. They are due to local interference with the maternal blood supply, owing to retroplacental hæmorrhage, thrombosis of the decidual sinuses or other causes. They are invariably found subtending retroplacental clot or in the neighbourhood of intraplacental hæmorrhages, and are found associated clinically with states of pregnancy toxæmia such as eclampsia, albuminuria of pregnancy or retroplacental hæmorrhage. Nevertheless, red infarction does not always accompany toxæmic states, nor is there always clinical evidence of toxæmia in cases where red infarcts are found.

Red infarction, if sufficiently extensive, will kill the fœtus from relative placental insufficiency. But infarction of this extent is almost invariably accompanied by severe conditions such as extensive retroplacental hæmorrhage and separation of the placenta ("accidental hæmorrhage") or eclampsia. It is not so much the red infarction *per se* as its causes or accompaniments that kill the fœtus. The recognition of red infarction may therefore be of great importance, as pointing out the cause of fœtal death, in cases where a clinical history of the case is wanting.

Red infarcts, during this investigation, were not looked for in specially favoured placentas. Each placenta, labelled according to number, was placed in a tank of formalin solution; after it had lain there about two weeks it was taken out and examined according to the method described on p. 4. Not many examples can have been missed. All suspiciously dark areas, as well as obvious infarcts, were examined microscopically. The results are most interesting and have convinced me that red infarction is no chance accident, but is a special change associated with special conditions. The following is a brief analysis of the cases, showing the associated maternal and other placental states.

I.—*Macerated Fœtuses.*

7. Several intraplacental hæmorrhages, no albuminuria.
11. Retroplacental hæmatoma.
26. Accidental hæmorrhage, albuminuria.
44. Accidental hæmorrhage.
110. Albuminuria of pregnancy,
127. Albuminuria of pregnancy.
133. Accidental hæmorrhage, retroplacental hæmatoma, albuminuria.
192. Accidental hæmorrhage, albuminuria.
197. Retro-placental hæmatoma, albuminuria.
217. Albuminuria of pregnancy, intraplacental hæmorrhage.
228. Retroplacental hæmatoma, intraplacental hæmorrhage.
280. Accidental hæmorrhage, intraplacental hæmorrhage.
282. Accidental hæmorrhage.
287. Retroplacental hæmatoma, albuminuria.
291. Accidental hæmorrhage, intraplacental hæmorrhage.
335. Accidental hæmorrhage, retroplacental hæmatoma, albuminuria.
357. Retroplacental hæmatoma.
412. Accidental hæmorrhage retroplacental hæmatoma, albuminuria.
416. Syphilis; accidental hæmorrhage, retroplacental hæmatoma, albuminuria.

II.—*Fresh Fœtuses.*

13. Accidental hæmorrhage, albuminuria.
28. Eclampsia.
48. Eclampsia.
160. Excessive red infarction; induction of labour for premature rupture of the membranes, ? albuminuria.
167. Accidental hæmorrhage.
191. Accidental hæmorrhage, albuminuria.
277. Accidental hæmorrhage (Cæsarean section), albuminuria.
286. Eclampsia, death.
293. Accidental hæmorrhage, albuminuria.
338. Placenta prævia. ? Albuminuria.

379. Placenta prævia. ? Albuminuria.
401. Normal breech labour, no antepartum hæmorrhage, no retroplacental hæmatoma, no albuminuria.

Amongst the macerated fœtuses all occurred in association with toxæmia of pregnancy states; 10 of accidental hæmorrhage, five of retroplacental hæmatoma, three of albuminuria of pregnancy and one of intraplacental hæmorrhage (No. 7). In four other of these placentas there was also present intraplacental hæmorrhage (Nos. 217, 228, 280, 291). These figures supply good evidence of the association of red infarcts with toxæmia of pregnancy, and demonstrate the value of routine examination of the placenta in cases of fœtal death. Amongst the fresh fœtuses were eight examples of toxæmia of pregnancy; five of accidental hæmorrhage and three of eclampsia. Amongst the other four no such association was apparent. Unfortunately the urine was not examined in the two cases of placenta prævia. In the case of the induced labour (No. 160), the infarction was very extensive; there was no albuminuria or other symptom of pregnancy toxæmia, and the case is one of great pathological importance.

Retroplacental Hœmatoma.

Reference has already been made to this in the section dealing with "accidental hæmorrhage." Here it may be repeated that in some cases of accidental hæmorrhage an old blood clot is found occupying a depression on the maternal surface of the placenta, showing that the extensive separation of the placenta which gives rise to accidental hæmorrhage is sometimes a slowly-developing process (Fig. 15). By a case of "retroplacental hæmatoma" I mean a case where the separation of the placenta has been slight and has not progressed, where there is no clinical manifestation of accidental hæmorrhage, and where the nature of the case is only revealed after the placenta has been examined (Fig. 17).

A blood-clot, of varying size, sometimes old and laminated, sometimes of more recent age, is found occupying a depression on the maternal surface of the placenta. There were five such cases in the present series; all were macerated (Nos. 11, 197, 228, 287, 357). If all the 300 placentas had been available for examination, it is probable that others would have been discovered. The fœtuses in these cases die from "relative placental insufficiency"; they are generally retained after death for a sufficient time to become macerated. Retroplacental hæmatoma is of course "concealed accidental hæmorrhage" in miniature. There are no maternal symptoms; the fœtus just dies. The discovery of these hæmatomata is another example of the great importance of examining the placenta in cases of fœtal death. There is usually albuminuria in the mother; albumin was found in two of the four cases in which the urine was examined. All were associated with red infarction and in one there was an intraplacental hæmorrhage (No. 228).

Intraplacental Hæmorrhage.

Six examples of intraplacental hæmorrhage were found (Fig. 18). These also were all associated with maternal toxæmia states. Two were from cases of albuminuria of pregnancy (Nos. 190, 217); two were from cases of accidental hæmorrhage (Nos. 280, 291); one was associated with retroplacental hæmatoma (No. 228). One (No. 7) was a very remarkable case of habitual fœtal death, in which there were multiple intraplacental hæmorrhages; there were no clinical signs of toxæmia (the case was kept under careful observation throughout the whole of pregnancy); but in the succeeding pregnancy, the patient had severe toxæmia with eclamptic convulsions. With the exception of this case, it is probable that none of the hæmorrhages were of sufficient magnitude to cause fœtal death from "relative placental insufficiency." But the condition is one more example of the extreme importance of routine examination of the placenta; it gives a clue to the accompanying clinical condition and is of especial value in cases where the clinical history may be wanting.

Microscopical examination of the placenta at the edges of the hæmorrhages reveals the fact that they are surrounded almost everywhere by decidual cells. They are, I believe, diffuse hæmorrhages originating from the decidual septa of the placenta. This mode of origin is of great interest in throwing light on the mode of origin of retroplacental hæmorrhage, for the two varieties have so many points in common that an analogy between the two is irresistible.

White Infarction.

The frequency of white infarcts, as well as the class of case in which they occur are well enough known. They are the late stage of local placental death, of which red infarction is the early. Of the 192 placentas examined, white infarcts were absent in 65 (38 fresh and 27 macerated fœtuses). There were six cases of excessive white infarction.

No. 44.—A case of accidental hæmorrhage. The umbilical cord contained one artery and the left hypogastric artery of the fœtus was absent. The placenta contained two large irregularly ovoid areas of white infarction, occupying about one-sixth of the volume of the placenta. Advanced hyaline degeneration of the villi was almost universal in the uninfarcted area. There were some late red infarcts. In this case the cause of fœtal death must be assigned to "relative placental insufficiency."

No. 127.—A case of albuminuria of pregnancy. The placenta contained 10 white infarcts.

No. 225.—This is a very remarkable example of the most extensive white infarction (Fig. 14). The infarct area occupied about two-thirds of the volume of the placenta. A catheter specimen of the maternal urine, taken a few hours after the normal labour, contained 0·2 per cent. of albumen. The patient was a primigravida. Auscultation of the fœtal heart-beat

had showed it to be living one hour before birth; at birth the heart was not beating. This is a clear case of death from "relative placental insufficiency." The small amount of healthy placental tissue sufficed for the requirements of the fœtus during the last part of pregnancy, and during labour at least until the last hour.

No. 297.—A case of albuminuria with uræmic convulsions. The placenta contained eight small white infarcts, not sufficient volume of the placenta was destroyed to cause "relative placental insufficiency."

No. 372.—A case of albuminuria of pregnancy. The placenta contained 12 small white infarcts and one larger one ("the size of a halfpenny"). Not enough destruction of placenta to cause "relative placental insufficiency."

No. 322.—Fœtus macerated, 2,350 grms. Placenta unusually small, weight-ratio 10·7. The edge of the placenta, in almost the whole circumference, was replaced by a zone of white necrosis measuring about 1 inch in width. Classed as case of relative placental insufficiency due to abnormally small placenta.

Unusual smallness of the Placenta as a cause of Fœtal Death.

It is reasonable to suppose that the fœtus requires a minimum amount of healthy placental tissue to maintain its life: in cases where this amount is not available, we may speak of "relative placental insufficiency." Cases of destruction of large areas of placenta by white or red infarction, and cases of separation of areas of placenta by retroplacental clots are all examples of relative placental insufficiency. By analogy with such cases it is reasonable to indulge in the hypothesis that a placenta may be relatively too small, though otherwise normal, to maintain the life of the fœtus; furthermore, the smaller the placenta relative to the needs of the fœtus, the smaller is the margin of safety against such events as infarction or separation, or against an excessive degree of the degenerative changes (occlusion of blood-vessels, degeneration and atrophy of the villi) which occur normally in the placenta as the full term of pregnancy is approached.

The hypothesis of unusual smallness of the placenta as a cause of fœtal death will become a theory if cases of fœtal death are found in association with an unusually small placenta, and if, in these cases, no other cause of death can be found.

Ten such cases have occurred in this series of 300 dead fœtuses (Nos. 30, 34, 184, 185, 273, 278, 322, 323, 371, 387). The large table of cases can be referred to for their more important details. It is a striking fact that 10 out of a total of 17 unusually small placentas should have belonged to macerated fœtuses for which no other cause of death was apparent.

All the fœtuses but one are 47 cms. or more in length, so the cause operates late; whether it is due to arrest of growth of

the placenta, or to a placenta small from the beginning, is impossible to say.

What I have put forward here is more in the nature of a suggestion than of an assertion. The idea of unusual smallness of the placenta as a cause of fœtal death must await further work for confirmation or negation.

Absence of one Umbilical and Hypogastric Artery.

In five macerated fœtuses (Nos. 32, 44, 60, 348, 412) and one fresh (No. 301), one umbilical artery and the corresponding hypogastric artery were wanting. The umbilical artery was in all cases absent throughout the whole length of the cord; and the corresponding hypogastric artery also in its whole length, from its origin from the iliac artery to the umbilical opening. Unfortunately, in the earlier cases of the series the defect was only looked for in those cases where the placenta and cord were available for examination; it was the absence of an artery from the cord that drew attention to the hypogastric arteries of the fœtus. Absence of an artery can, of course, equally well be found by examining the stump of the cord on the fœtus. I can find no information on this anomaly in anatomical or obstetrical literature, except in the report of a case of fœtal deformity. ("Allantoid-Angiopagous Twins," D. M. C. Dougal, *Journ. Obstets. and Gyn. of Brit. Emp.*, 1914.) To find out if it was a common one, and found in other than dead fœtuses, I examined the placentas and cords of 450 infants born at the City of London Maternity Hospital and found only one artery in two cases.

In the case of the dead fœtuses, absence of an umbilical artery was always accompanied by absence of the corresponding hypogastric artery.

The circumstances of its occurrence according to my observations are, therefore :—

> In the cords of 450 living infants, there was only one artery in two cases.
>
> In the cords of 94 fresh fœtuses, there was only one in one case.
>
> In the cords of 98 macerated fœtuses, there was only one in five cases.

The preponderance of macerated fœtuses naturally leads to the enquiry whether the defect leads to deficient fœtal circulation in the placenta and, directly or indirectly, to a relative placental insufficiency. I believe its association with fœtal death is more than a coincidence; but much more observation is required. It is not unreasonable to suppose that, if one hypogastric artery is absent, the volume of blood flowing into the placenta is reduced by one-half (the remaining umbilical and hypogastric artery did not appear to be hypertrophied in any of the cases); the placenta would then develop only half its efficiency and the margin of safety for the fœtus would be reduced dangerously.

In such a case if the placental tissue remained comparatively healthy, all would be well; but if there occurred only a moderate degree of infarction, or if normal degenerative changes were in excess, or if the placenta were an abnormally small one, there would be a danger of "relative placental insufficiency." The last consideration might explain why some fœtuses with this defect live, whilst others die. In the two cases of living children, and in the one case of fresh dead fœtus, the placentas were free from infarction, but in the cases of the five macerated fœtuses, all the placentas showed defects. Interesting developmental points are raised as to the influence of the defect on the growth and vascularisation of the chorionic villi. We can now turn to the results of the microscopical examination of the placentas. Unfortunately, not more than two blocks of tissue from each placenta were examined. In three placentas (Nos. 44, 60 and 412) the villi were remarkable for the scantiness of the blood-vessels; in many the vessels were absent altogether. In Nos. 32 and 348, the vascularity was normal. In the placenta from one of the living infants, many of the villi were unusually small and avascular.

The details of the cases are as follows :—

No. 32.—Weight of fœtus, 3,032 grms.; length, unknown. The placenta was unusually small, the weight-ratio being 11. There were no infarcts. The villi contained the usual number of vessels. Right hypogastric artery absent. No discoverable cause for fœtal death.

No. 44.—Weight of fœtus, 970 grms.; length, 42 cms.; therefore fœtus was unusually light in proportion to its length. The fœtus had spina bifida and double talipes equinus. Clinically the case was one of accidental hæmorrhage. The base of the cord was expanded and contained a piece of liver. The placenta was of normal size, its weight-ratio being 5·5. It contained an unusual number of white infarcts, and there was a diffuse hæmorrhage beneath the chorionic membrane. Microscopical examination showed a large number of villi without blood-vessels. The left hypogastric artery was absent.

No. 60.—Weight of fœtus, 2,850; length, 51; the fœtus was therefore light in proportion to its length; it was also hydrocephalic. The placenta was of normal size, the weight-ratio being 7·5. There was a remarkable absence of vessels in the villi; hyaline degeneration was in excess. The umbilical vein was expanded at the base of the cord and contained a large white ante-mortem clot. The left hypogastric artery was absent; it was present as a tiny vessel for the first inch from its origin from the iliac artery, but for the rest of its course was a tiny strand.

No. 348.—Weight of fœtus, 1,080; length, 35. Placenta slightly below normal size. No infarcts; but excessive hyaline degeneration of the villi. Left hypogastric artery absent.

No. 412.—Weight of fœtus, 3,120; length, 52. The case was one of accidental hæmorrhage and has been classified as such. The placenta was of normal size, the weight-ratio being 7. There was much red infarction.

From the evidence at hand, I have assigned the cause of death in cases No. 32, 60 and 348 to absence of the artery, there being no other known cause for fœtal death, combined, in No. 32 with an abnormally small placenta, in No. 60 with avascularity of the villi, and in No. 348 with excessive hyaline degeneration of the villi. Further work must test the correctness of these views; as expressed here, they will at all events serve for a standard to work against.

VIII.—(4) The Syphilitic Placenta.

All modern text-books describe certain changes in the placenta in cases of fœtal syphilis. Though the individual changes may be well enough known, we are without precise information as to how often these changes are found and to what extent they are pathognomonic of syphilis. It is necessary to have a clear idea of the diagnostic worth of these changes: for we may sometimes wish to use the placenta as a means of diagnosing fœtal syphilis when other means, such as the presence of spirochætes in the fœtal tissues or fœtal osteochondritis, are not available.

The changes commonly described in the syphilitic placenta may be divided into three: (*a*) naked-eye appearances; (*b*) the ratio of the weight of the placenta to the weight of the fœtus; and (*c*) the histological changes. Examination of the placenta for the spirochæta pallida has already been discussed on p. 29.

The number of syphilitic placentas available for investigation is 34, comprising 30 from the spirochæte-positive and 4 from the spirochæte-negative fœtuses.

(*a*).—*Naked-Eye Appearances.*

In my experience even the most careful naked-eye examination of the syphilitic placenta reveals nothing of note; there is nothing to distinguish it from many other placentas. Syphilis cannot, therefore, be diagnosed from the external appearance or cut surface of the placenta. It is important to emphasize this because, even nowadays, there is a great deal of loose talk about the appearance of the syphilitic placenta. One hears or reads such statements as: "The placenta was from its appearance obviously syphilitic." The appearances which are wrongly supposed to denote syphilis are voluminous cotyledons, deep sulci, pale placental tissue, grey and greasy maternal surface, fibrous areas, unusual softness and friability. Some of these appearances may occasionally be seen in syphilitic placentas, but they are just as commonly found in other conditions. Nor, as is sometimes stated, are white infarcts commonly found. On the contrary, in this series, the placentas were remarkably free

from infarcts. In 30 placentas from spirochæte-positive fœtuses, sliced through at intervals of a quarter of an inch, white infarcts were only found in two; No. 41 contained two and No. 227 one small infarct. In the remainder it was specially noted that there were no infarcts. In No. 416 there was a considerable area of red infarction, associated with retroplacental clot; this was a case of accidental hæmorrhage.

(*b*) *The Weight-Ratio.*

The experience gained by the investigation of this series of cases shows that far too much importance has been attached to the increased weight of the syphilitic placenta. In Table VIII. are shown the mean ratios for syphilitic placentas: (*a*) the mean for all cases; (*b*) the mean when the fœtuses are divided into weight-groups.

In Table IX. are displayed all together the ratios for fresh and macerated non-syphilitic, and for syphilitic fœtuses, both for total fœtuses and for fœtuses divided into weight-groups. From examination of this Table the mean ratios of syphilitic and non-syphilitic fœtuses can be contrasted; it will be noted that the mean ratio for syphilitic is lower than that for non-syphilitic, but that the difference is not great.

The real test of the worth of heaviness of the placenta as a sign of syphilis is best got by arranging the ratios in tables showing their frequency distribution amongst fœtuses of different weights. This will be found in Table X.; it is at once apparent that a considerable number do not show heaviness, their ratios being equal to or greater than the mean ratios for the non-syphilitic placentas. From the Table can be picked out six placentas, belonging to fœtuses of less than 1,800 grms., with a ratio of six or more (six being the mean ratio for non-syphilitic fœtuses of this weight), and six, belonging to fœtuses of over 1,800 grms., with a ratio of eight or more (eight being the mean ratio for non-syphilitic fœtuses of this weight). That is, out of 34 syphilitic placentas, only 22, or 65 per cent., were heavier than normal. This shows that heaviness of the syphilitic placenta is not constant.

When the ratios of the non-syphilitic placentas shown in Table X. are scrutinised, it will be seen that a number are heavy, their ratios being equal to or greater than the mean ratio for the non-syphilitic placentas. These heavy placentas have already been pointed out and discussed in detail on p. 102. From the foregoing it is sufficiently obvious that the quality of heaviness is not constant in the syphilitic placenta, and is commonly met with in non-syphilitic placentas; heaviness of the placenta is, therefore, of scarcely any value as a diagnostic sign of syphilis.

III.—*Histology of the Syphilitic Placenta.*

Of the 37 fœtuses whose tissues contained spirochætes the placentas were examined microscopically in 26. The histological

points which have been noted as characteristic of syphilis, but which were not found in all of these 26 placentas, are :—

(1) A uniform increase in the size of the villi, resulting in their being more crowded together than normally and implying a corresponding reduction in the size of the intervillous spaces. This change is due to an increase in the amount and density of the stroma, the collagen fibres being more numerous and the cells more abundant and closely packed.

(2) A greatly diminished vascularity of the villi. The blood-vessels are usually absent; when present they are greatly diminished in number and size. The walls of the vessels, when present, are not altered; they still appear as fine endothelial-lined channels in the stroma.

These changes (Fig. 20) sometimes affect the whole placenta uniformly, and sometimes only partially. The villi coming from one or more main chorionic stems may be affected, whilst others escape, healthy and changed villi being seen side by side in the same section. It is obviously absurd to diagnose syphilis from the discovery in a section of a few isolated large, dense, avascular villi. Furthermore, it is obvious that little help may be got from one section; either one very large section or several small ones must be used.

So far as my observations have gone I have not found special changes in the blood-vessels of the placenta. It is commonly stated that the vessels of the villi in syphilis are obliterated by an endarteritis. I have never observed this. What vessels there are reveal themselves as little circular collections of endothelial cells, their walls are not increased in thickness nor have the endothelial cells multiplied.

The explanation of the avascularity of the villi in syphilis, I believe, is that the terminal villi have never been properly vascularised by the downgrowth of blood-vessels, and not that the villi have become devascularised by a later obliteration of the vessels.

I have one other point to mention—the occurrence of a small round-celled infiltration in the stroma of the villi, indicating an inflammatory change. In a few sections, at long intervals, I found places where neighbouring villi had coalesced and where the stroma was infiltrated with small round cells. These appearances were exceptional and must await further observation.

In five cases (Nos. 21, 53, 176, 328 and 377) I found a little node composed of leucocytes, three times lying in the thick chorionic membrane and twice in the decidua. They all occurred in syphilitic placentas. If these were tiny gummata, they would be of material aid in diagnosing placental syphilis (Figs. 23 and 24).

According to the completeness with which these changes were found in the 26 syphilitic placentas I have examined, the placentas were grouped into three classes: (1) typical, (2) suspicious, (3) negative or normal. When the changes are perfectly

developed and uniformly distributed they are easily recognisable. But where the distribution of enlarged villi is patchy, or where the blood-vessels are only moderately diminished, the diagnosis is extremely difficult and it is only safe for those who are in the habit of regularly examining placental sections to give an opinion. For the changes do not consist in the formation of foreign tissue elements, or of rearranged structure; they are simply exaggerations of the normal, and it is sometimes difficult to decide between what is only a physiological abnormality and a really pathological change. The two are not separated by a sharp line. A series of control sections is of great assistance.

A " typical " section is one where all the villi are affected as regards increase in size, density of the stroma, avascularity and close packing. The typical appearance of such a section is shown in Fig. 20.

In a " suspicious " section, the change is a patchy one; there are groups of enlarged avascular villi, but the groups are neither so large nor so numerous, nor are the villi so avascular as in the standard typical section (Fig. 21).

Fig. 22 is a section of the placenta in a case of general œdema of the fœtus; the villi are uniformly enlarged but are not dense and avascular, the stroma being loose and œdematous. Contrast this with Fig. 20.

Results of the histological examination of the 26 placentas belonging to spirochæte-positive fœtuses are shown in Table XI.; typical changes were found in 61 per cent., which is sufficient evidence that the change is not constant.

TABLE XI.

Placentas of 26 *Spirochæte-positive Fœtuses.*

Typical of Syphilis in	16, or 61 per cent.
Suspicions in	6, or 23 ,,
Negative in	4, or 15 ,,

Placentas of 91 *Spirochæte-negative Fœtuses.*

54 *Macerated Fœtuses.*		37 *Fresh Fœtuses.*	
Typical	5	Typical	0
Suspicious	6	Suspicious	4
Normal	43	Normal	33

SUMMARY AS REGARDS AGREEMENT OF SECTIONS WITH DIAGNOSIS OF SYPHILIS.

117 *Placentas.*

Typical, 21	Undoubted fœtal syphilis in 19 (90 per cent.).
Suspicious, 16	,, ,, 6 (38 ,,).
Normal, 80	,, ,, 4

In the same Table are shown the results obtained from the examination of the placentas of 91 spirochæte-negative fœtuses. It is seen that five of these placentas showed typical, and

10 suspicious changes. The details of the cases from which these sections came may now be examined in detail, and the results give strong support to the reliability of the placental histology for the diagnosis of syphilis.

There are five "typical" sections amongst the macerated spirochæte-negative fœtuses (Nos. 12, 65, 124, 210 and 335). The first three of these prove to be undoubted cases of fœtal syphilis; all had maternal positive Wassermann reactions; two of the fœtuses showed osteochondritis. The third fœtus showed no osteochondritis, but I had the mother under treatment before pregnancy, during which time the Wassermann reaction became negative. The two remaining cases showed no other evidence whatever of syphilis; one was a case of accidental hæmorrhage and albuminuria (No. 335); in the other (No. 210) the cause of fœtal death was undetermined. The results obtained from "typical" sections can be thus summarised. In the microscopical examination of 117 placentas, 21 gave appearances typical of syphilis; of these 21, 19 were undoubted cases of syphilis, whilst in two there was no other evidence of it. From this small series it can be stated that the diagnosis of syphilis from a typical section will be correct in 90 per cent. of cases. Thus, a typical section is highly reliable.

I will next consider the 10 "suspicious" sections, amongst the spirochæte-negative fœtuses. In one (No. 163) the Wassermann reaction was positive in the mother; the fœtus was killed during a breech-labour by cerebral hæmorrhage; there was no other evidence of syphilis and the case has not been classed as such. Two (Nos. 87, 365) were cases of general fœtal œdema. An interesting point comes out in the remaining seven (Nos. 38, 133, 190, 198, 309, 357, 389); all were cases of toxæmia of pregnancy—accidental hæmorrhage or albuminuria of pregnancy, or both. The results forthcoming from "suspicious" sections can be thus summarized: in the microscopical examination of 177 placentas, 16 gave appearances "suspicious" of syphilis; of these 16, six are undoubted syphilis. From this small series it can be stated that the diagnosis will be correct in only 38 per cent. A merely suspicious section is unreliable.

SECTION IX.

Addenda.

(1) List of Cases with Cause of Death Undetermined.

(2) Maternal Deaths in the Present Series.

IX.—(1) List of Cases with Cause of Death Undetermined.

In the case of seven fresh fœtuses the cause of death was not discoverable.

No. 55.—Premature fœtus, 38 cm. long; normal labour; fœtus appeared extremely anæmic, all organs pale; all organs normal in weight; histological examination not made; placenta normal in size and appearance, but not examined histologically. The mother had had four former premature labours at about same date as this one, and none of the children lived more than a few hours.

No. 75.—Induction of labour at 36th week for contracted pelvis, but there was no evidence of an obstetrical cause of death. Slight fœtal ascites; kidneys and thymus larger than normal. Placenta normal. Fœtus died early in labour.

No. 107.—Mature fœtus; normal labour; no abnormal post mortem findings; all organs of normal weight. *Placenta not examined.*

No. 298.—Moderately premature fœtus, 46 cm. long. Normal labour. No abnormal post mortem findings. *Placenta not examined.*

No. 347.—Mature fœtus, normal labour. A slight film of hæmorrhage over cerebral hemispheres, but not enough to cause death; petechial hæmorrhages over pleural surface of lungs; a few subcapsular hæmorrhages in liver; petechial hæmorrhages over visceral pericardium. "Acute anaërosis" of fœtus, cause unknown. *Placenta not examined.*

No. 352.—Mature fœtus, the second of twins; other twin born alive. Born before arrival of midwifery attendant. No abnormal post mortem findings. *Placenta not examined.*

No. 405.—Premature fœtus, length 36 cm. Normal vertex labour; child died during labour. No abnormal post mortem findings. *Placenta not examined.*

Amongst the macerated fœtuses were 25 where the cause of death could not be determined.

No. 18.—Mature fœtus; no maternal disease, no complications of pregnancy. *Placenta not examined.*

No. 25.—Premature fœtus, length 39 cm.; XI. para., only one other dead-birth; no maternal disease, no complications of pregnancy. *Placenta not examined.*

No. 68.—Moderately premature fœtus. Maternal Wassermann positive. No complications of pregnancy. No signs of syphilis in fœtus or placenta and evidence not considered sufficient to justify diagnosis of fœtal syphilis. "Possible syphilis."

No. 122.—Moderately premature fœtus. Mother has epilepsy, but no complications of pregnancy. Hæmorrhage into left suprarenal capsule. *Placenta not examined.*

No. 164.—Premature fœtus. Length, 36 cm. Maternal Wassermann positive. No complications of pregnancy. No signs of syphilis in fœtus. Evidence not considered sufficient to justify diagnosis of syphilis. *Placenta not examined.* "Possible syphilis."

No. 150.—Mature fœtus. No maternal disease, no complications of pregnancy. Spleen, kidneys, and suprarenals below normal weight. *Placenta not examined.*

No. 152.—Premature fœtus, length 40 cm. No maternal disease, no complications of pregnancy. Placenta normal; liver, spleen and suprarenals unusually large.

No. 189.—Premature fœtus, length 40 cm. No maternal disease, no complications of pregnancy. *Placenta not examined.*

No. 211.—Premature fœtus, length, 41 cm. No complications of pregnancy. Maternal Wassermann positive. No signs of fœtal syphilis. Placenta normal. Evidence not sufficient to justify diagnosis of syphilis. "Possible" syphilis.

No. 215.—Mature fœtus. No maternal disease, no complications of pregnancy. Placenta normal.

No. 219.—Premature fœtus. Length, 36 cm. No complications of pregnancy. *Placenta not examined.*

No. 221.—Mature fœtus. No complications of pregnancy. Placenta normal.

No. 224.—Small fœtus, one of triplets. The first was born alive; the other two were macerated, their lengths and weights being 28 cm., 45 cm., and 350 grm. and 1,570 grm. respectively. *Placenta not examined.* No maternal disease and no complications of pregnancy.

No. 238.—A very large mature fœtus; no complications of pregnancy or maternal disease; moderate hydramnios; no placental abnormality; VIII. para., no former dead-births.

No. 239.—Mature fœtus. No pregnancy complications. *Placenta not examined.*

No. 270.—Premature fœtus, length, 42 cm. No maternal disease or pregnancy complications. Spleen below standard weight. *Placenta not examined.*

No. 319.—Mature fœtus; kidneys slightly below normal weight. No maternal disease or pregnancy complications. *Placenta normal.*

No. 321.—Premature fœtus, length, 37 cm. No pregnancy complications. No maternal disease. *Placenta not examined.*

No. 336.—Premature fœtus, length, 35 cm. No pregnancy complications or maternal disease. *Placenta not examined.*

No. 376. Moderately premature fœtus. No pregnancy complications or maternal disease. *Placenta not examined.*

No. 384.—Mature fœtus. No pregnancy complications or maternal disease. Suprarenals and spleen above normal size. *Placenta not examined.* XI. para., no former dead-births.

No. 385.—Premature fœtus, one of twins, the other living, length 37 cm. No maternal disease or pregnancy complications. Placenta not examined. X. para., no former dead-births.

No. 388.—Mature fœtus. Spleen and kidneys slightly below standard weight. No maternal disease or pregnancy complications. *Placenta not examined.*

No. 394.—Premature fœtus, length 38 cm. Suprarenals slightly below standard weight. No maternal disease or pregnancy complications. Placenta normal.

No. 407.—Moderately premature fœtus. No maternal pregnancy complications. (The mother died from puerperal sepsis.) Placenta normal.

The fact that in 32 (11 per cent.) of the 300 cases a cause of death could not be determined invites criticism. No doubt, this is chiefly due to gaps in our knowledge of the subject; as more becomes known about the physiology and pathology of the fœtus and placenta, and of pregnancy and labour, this class of case will shrink.

It must be confessed that imperfect investigation accounted for a large proportion. In no less than 19 (five fresh and 14 macerated) the placenta was not examined, because it was not available. This was a serious omission, as has been emphasized in the section on the "placental causes of fœtal death." Everyone who supplied me with material was repeatedly reminded I must have the placenta. In the in-patient hospital cases I seldom failed to get it; but in the district cases the placenta was apt to be summarily destroyed before it could be rescued. In certain cases the maternal urine was not examined; this, again, only in district cases. The mothers were seldom under routine antenatal care. But for these two omissions, there can be little doubt that the number of cases with "cause undetermined" would have been less. This class of case

demonstrates the importance of omitting nothing in the investigation of the mother, fœtus and placenta. It also demonstrates the need for further research into the fundamental principles of this subject.

IX.—(2) Maternal Deaths.

Eight mothers died during or just after labour :—

No. 281.—Severe broncho-pneumonia; spontaneous labour at the 28th week; died the following day.

No. 367.—Accidental hæmorrhage; died from collapse two hours after delivery.

No. 403.—Severe albuminuria of pregnancy, twins. Induction of labour with bougies. The patient got a very severe antepartum infection, and died four days after labour.

No. 407.—Neglected transverse presentation, macerated fœtus. Arrived in hospital with severe antepartum sepsis; easy delivery by traction on breech; death two days afterwards.

No. 48.—Eclampsia; patient died a few hours after delivery.

No. 159.—Accidental hæmorrhage; patient died a few hours after delivery.

No. 261.—Placenta prævia; patient died from septicæmia a few days after delivery.

No. 331.—Contracted pelvis, obstructed labour. Delivery after perforation of head. Rupture of uterus. Septic peritonitis, death a few days after labour.

There, of course, may have been other maternal deaths later in the puerperium; but I have no information as I did not follow up the subsequent maternal history, except in the case of patients at the City of London Maternity Hospital.

SECTION X.

Conclusions.

(1) The Chief Causes of Fœtal Death.

(2) The Prevention of Fœtal Death.

X.—(1) The Chief Causes of Fœtal Death.

The main conclusion is seen in Table I., which shows the distribution of the primary causes of death. From the mass of detail accumulated during this investigation certain generalizations can be made. To begin with, I feel that this investigation has been on general lines, and that I have only been able to go over the ground more or less superficially. Such knowledge as is available on the subject has been made use of. The enquiry into syphilis has been made with great precision. There are many gaps in our knowledge of the fundamental points necessary for the prosecution of a more complete enquiry; many of these are of an elementary nature and can be settled by patient spade-work on a large material. An example is the histology of the fœtal organs, which at present it is almost impossible to interpret aright, because so little is known of the normal and variations of the normal. Another example is the placenta; extremely little work has been done on abnormal placental states, and in placental histology it is extremely difficult sometimes to decide between the normal and the abnormal. There is much knowledge already to hand, but it can be made so very much more precise. Another thing, of which we are as yet on the fringe, is bacterial infections of the fœtus, their relation to maternal antepartum infections, and the *rôle* played by the placenta in such cases.

Coming now to syphilis, the results got have been important. I have always thought that syphilis as a cause of fœtal death has been given an exaggerated prominence, and that precise work on a large material would unmask its pretensions. The Royal Commission on venereal diseases was unable to obtain any precise information, but had to rely on indirect evidence. The Commissioners were thereby led to give very great prominence to syphilis as a cause of "still-birth," although they could not state figures, and went the length of using the still-birth rate as an index of the prevalence of syphilis in general: "As syphilis is a frequent cause of still-birth, these figures" (*i.e.*, the statistics of still-birth) "have an important indirect bearing

on the question" (of the prevalence of syphilis amongst the general population—Section 26). In Section 28, they further state that "still-births occurring before the 28th week of pregnancy would not be notified and *must include a considerable number due to syphilis.*" Furthermore, the Medical Officer to the Local Government Board, in his Report for 1913–14, was led to believe, from information given him, that syphilis was responsible for 50 per cent. of still-births. Reference to my table showing primary causes of death shows that amongst the 300 fœtuses were 42 of proven syphilis, and 6 of "probable" syphilis; making an incidence of 16 per cent. There were a further 14 cases of "possible" syphilis; that is, of cases where the mothers' Wassermann reaction was positive but where there was no other supporting evidence in the fœtus or placenta. These results are in close accordance with those obtained by Dr. Whitridge Williams in the United States.* Another unexpected discovery has been the extreme frequency with which cranial injuries occur in fresh fœtuses. Out of the 167 fresh fœtuses—*i.e.*, those which died during labour—no less than 48 per cent. had tearing of the septa of the dura mater and cerebral hæmorrhage. The almost constant discovery of these injuries in fœtuses born dead after breech-delivery is a fact of great practical importance, and indicates the urgent necessity of teaching a better method of managing the delivery of the after-coming head than the one at present in vogue. I have gone very fully into the mode of origin of these injuries and have worked out a new theory of the functions of the dura mater septa, and of the mode of origin of cerebral hæmorrhage.

A generalization of great importance is that more fœtuses were killed by the complications of labour than died during pregnancy from maternal or placental disease. From the preventive point of view this is an observation of the highest importance. To begin with there were more fresh fœtuses (deaths during labour) than macerated (deaths during pregnancy); the respective numbers were 167 fresh and 133 macerated. A comparison between the number of deaths from the complications of labour, from maternal conditions, from fœtal defects and from placental states is best seen by placing them in percentage groups :—

	Per cent.
Complications of labour (including antepartum hæmorrhage and postmaturity of the fœtus)	51
Syphilis	16
Toxæmias of pregnancy	10
Chronic renal and other maternal diseases	2
Placental states leading to relative placental insufficiency	6
Fœtal deformities	5
Cause of death unknown	11

* Limitations and Possibilities of Prenatal Care. J. Whitridge Williams *Jnl. Amer. Med. Assoc.*, 1915. LXIV. 95.

X.—(2) The Prevention of Fœtal Death.

Although this report is supposed to deal only with the causation of fœtal death, I should like to end with a few remarks on its prevention; for, I take it that the chief aim and object of an investigation of this nature is to give a lead to the preventive aspect of the subject and to urge it forward. Of course, the total abolition of fœtal death is beyond all dreams; but that a large mass of it results from essentially preventable causes is sufficiently obvious. One point to settle is the relative frequency with which the several causes of death occur in a sample of cases; another is to decide what group of causes is most likely to yield to preventive attack.

I shall try and estimate how many of these fœtal deaths could have been prevented. Before this is considered in detail it may be stated that there are two distinct periods when causes of fœtal death act, when death of the fœtus occurs, and when preventive treatment can be carried out: (1) during pregnancy, the antenatal period; (2) during labour, the intranatal period. Antenatal methods are the strategy, intranatal methods the tactics, of obstetrics. There is, I believe, a tendency nowadays to exalt the strategy at the expense of the tactics. How wrong this unequal conception of midwifery practice is, becomes at once apparent when we realise that when treatment is indicated, as the result of antenatal examination, such treatment usually consists in the application of intranatal methods. The only purely antenatal treatment consists in the treatment of syphilis and of appropriate cases of toxæmia of pregnancy. Otherwise, treatment is by intranatal methods, such as the induction of premature labour, Cæsarean section, or the careful management of labour. Though antenatal work is of immense importance, yet the importance of the intranatal period and the necessity of getting a better standard of midwifery practice must be recognised much more than it is at present.

Bearing in mind these two periods—antenatal and intranatal—we can, for the purposes of preventive treatment, place cases of fœtal death into four groups:—

1. Those, the cause of which can be discovered during the antenatal period and can be prevented by purely antenatal treatment. The great representative of this group is syphilis. Another example is toxæmia of pregnancy, many cases of which can be kept within reasonable bounds, or even cured, by simple antenatal treatment. Still another example is breech presentation, which can be corrected by external version.

2. Those, the causes of which can be discovered during the antenatal period, but which depend for their prevention on intranatal methods. The best example of this is contracted pelvis. If discovered early enough in pregnancy the obstetrical treatment can be planned, and will be either waiting for natural labour, with the help of forceps if necessary, or induction of premature labour, or Cæsarean section. Another example is

afforded by those cases of toxæmia of pregnancy which do not yield to antenatal treatment and which must be treated by induction of premature labour. Another rare example is obstructive pelvic tumours.

3. Those, the causes of which are not discoverable in the antenatal period, but which make their presence known just before or coincidently with the onset of labour, or during the course of labour. Examples of this group which are prolific of fœtal death are the antepartum hæmorrhages. Other common ones are prolapse of the umbilical cord and cases of prolonged labour from such causes as persistent occipito-posterior presentations. In this group most of the fœtuses have cerebral hæmorrhage and tearing of the septa of the dura mater, consequent on delivery by forceps or version.

4. Those, of which the causes, so far as our present state of knowledge stands, are not discoverable or preventable by any means whatever. These include the great group in which the cause of fœtal death cannot be determined, the cases of relative placental insufficiency due to abnormal smallness of the placenta or to the destruction of a large area of placenta by infarction, those cases in which a blood-clot is found behind the placenta, and cases of severe fœtal deformity such as anencephaly, hydrocephalus, and œdematous states of the fœtus.

I will now attempt to make a rough estimate of how many fœtal deaths in the present sample would have been preventable, and will give a rough indication of the part played by antenatal methods alone, by combined antenatal and intranatal methods, and by intranatal methods alone.

Table.

——	Incidence.	Preventable.	Antenatal.	Ante- and Intra-natal.	Intra-natal.
	Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Syphilis	16	16	16		—
Toxæmia of pregnancy	10	6	3	3	—
Complications of labour (including antepartum hæmorrhage)	51	30	1	9	20
Chronic renal and other maternal diseases	2	1	1	—	—
Cause undeterminable	11	0	—	—	—
Relative placental insufficiency and retroplacental clot	6	0	—	—	—
Fœtal deformities	5	0	—	—	—

It is thus conceivable that about half the fœtal deaths are preventable; that of these, about 20 per cent. (of which three-quarters are due to syphilis) could be prevented by antenatal methods alone, about 12 per cent. depend for their prevention on combined antenatal and intranatal methods, and about 20 per cent. on intranatal methods alone.

The foregoing line of reasoning leads to the conclusion that ante- and intra-natal methods are very closely correlated in preventive work, and that the most promising groups for attack are: (1) the complications of labour; (2) syphilis; (3) the toxæmias of pregnancy—in order of the number of successes likely to be got.

The treatment of the complications of labour falls to the doctor, as opposed to the midwife. The improved teaching of midwives has come, the establishment of antenatal centres has come and is growing. But the most urgent reform of all, the improved training of doctors, has yet to come. Fœtal mortality is not likely appreciably to fall until their standard of midwifery skill is raised.

APPENDIX.

TABLE XII.—TABLE OF CASES WITH THEIR IMPORTANT DETAILS: FRESH FŒTUSES.

TABLE XII.—TABLE OF CASES WITH THEIR IMPORTANT DETAILS: MACERATED FŒTUSES.

TABLE XIII.—LIST OF WEIGHT-RATIOS FOR THE FŒTAL ORGANS IN ALL CASES.

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Abbreviations used in Table of Cases.

L. = Liver.
S. = Spleen.
K. = Kidney.
SR. = Suprarenal.
T. = Thymus.
Th. = Thyroid.
Sp. = Spirochæta pallida.
W.R. = Wassermann reaction.
N.S. placenta = Normally situated placenta.
A.P.H. = Antepartum hæmorrhage.
N.E. = Not examined.

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
13	55	4,082	M.	—	Acute anaërosis, L—, SR.—.	—	A.P.H., accidental; albuminuria.	535	7·4	Red infarction	Separation of N.S. placenta.	Rel. plac. insuffic.
17	45	1,960		—	No signs of acute anaërosis, S.—.	—	A.P.H., placenta prævia	—	—	N.E. - -	Placenta prævia	Probably fœtal anæmia.
19	42	2,000		—	No signs of acute anaërosis.	—	A.P.H., placenta prævia	254	7·8		Placenta prævia	?
27	50	3,146	F.	—	Slight cerebral hæmorrhage, signs of acute anaërosis.	—	A.P.H., accidental -	—	—	N.E. - -	Separation of N.S. placenta.	Acute anaerosis.
28	40	1,650	M.	—	Necrosis of cells of liver, kidney, and suprarenals, K—, SR—.	—	Eclampsia; Cæsarean section.	270	6·1	Red infarction	Eclampsia -	Fœtal toxæmia.
33	52	3,175	M.	—	Torn tentorium; cerebral hæm.	—	Contracted pelvis; forceps.	—	—		Contracted pelvis	Cerebral hæm.
35		3,968		—	Double cystic kidneys, one ruptured; extensive cranial injuries, T—.	—	Labour obstructed by large size of fœtal abdomen.	440	9	N.E. - -	Deformity of fœtus, double cystic kidneys.	Fœtal injuries.

I

TABLE XIIa.—FRESH—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
36		1,700	M.	—	Slight cerebral hæmorrhage; acute anaërosis.	—	A.P.H., accidental; albuminuria.	—	—		Separation of N.S. placenta.	Acute anaërosis.
42	43	1,672	M.	—	Torn tentorium; cerebral hæm, L+.	—	Breech labour - -	350	4·7	Normal - -	Breech labour -	Cerebral hæm.
45	56	4,110	M.	—	Torn tentorium; cerebral hæm.	—	Forceps; prolonged labour; persist. O.P.	—	—		Prolonged second stage.	Cerebral hæm.
46			F.	—	Slight cerebral hæm.; signs of acute anaërosis.	+	A.P.H., accidental -	320	—	Normal - -	Separation of N.S. placenta.	Acute anaërosis.
47	50	3,458	F.	—	Slight cerebral hæm.; signs of acute anaërosis, K+.	—	A.P.H., accidental -	—	—	N.E. - -	Separation of N.S. placenta.	Acute anaërosis.
48	41	1,302	F.	—	Necrosis of cells of liver and kidney; acute anaërosis, T—.	—	Eclampsia; died - -	700	1·8	Retroplacental hæm.; red infarction.	Eclampsia -	Rel. plac. insuffic.
51	45	2,600	M.	—	Torn tentorium; cerebral hæm., T+.	—	Forceps; prolonged labour; persistent O.P.	—	—		Prolonged 2nd stage.	Cerebral hæm.
55	38	1,100	M.	—	Organs pale, no histolog. exam.	—	Repeated premature labours.	204	5·4	N.E. - -	Unknown.	
57	50	2,400	M.	—	Torn tentorium and falx; cerebral hæm., K+.	—	Breech labour - -	—	—		Breech labour -	Cerebral hæm.
64	44	2,300	F.	—	Signs of acute anaërosis	—	Albuminuria - -	—	—	N.E. - -	Albuminuria of pregnancy.	Acute anaërosis.
75	52	2,820	M.	—	Slight fœtal ascites, K+, SR—, T+.	—	Contracted pelvis; induction of labour.	300	9·4	Normal - -	Unknown.	
76	42	1,750	M.	—	Hæmatoma of falx; signs of acute anaërosis.	—	A.P.H., accidental -	—	—	N.E. - -	Separation of N.S. placenta.	Acute anaërosis.
82	52	3,000	M.	—	Torn tentorium; cerebral hæm., SR—.	—	A.P.H., placenta prævia; version.	260	11·5	Normal - -	Placenta prævia	Cerebral hæm.

87	52	3,340	M.	—	Torn tentorium and falx; cerebral hæm.; general œdema, S+, K−, SR−, T−.	—	Breech labour - -	600	5·5	Large irregular villi; "suspicions" of syphilis.	General œdema.	
103	40	2,450	F.	—	Torn tentorium; cerebral hæm., SR+.	—	A.P.H., placenta prævia	330	7·4	Few white infarcts.	Placenta prævia	Cerebral hæm.
107	50	3,150	M.	—	No cerebral hæm.; no signs of acute anaërosis, SR−.	—	Normal labour - -	—	—	N.E. - -	Unknown.	
108	52	3,320	F.	—	Torn tentorium; fracture of parietal bone; cerebral hæm.	—	Albuminuria; induction of labour; prolapsed cord; forceps.	560	5·9	Normal - -	Albuminuria of pregnancy.	Prolapsed cord; cerebral hæm.
111	—	3,320	F.	—	Torn tentorium; cerebral hæm, SR+.	—	Premature rupture of membranes; bag; transverse present., version.	400	8·3	- - -	Transverse present.	Cerebral hæm.
113	36	1,040	F.	—	Nothing of note - -	—	Breech labour, premature.	210	4·9	Normal - -	Prematurity.	
116	45	2,050	M.	—	Fœtus was bloodless; no cerebral hæm., SR−, T+.	—	A.P.H., placenta prævia	—	—	Normal - -	Placenta prævia	Fœtal anæmia.
123	36	1,010	M.	—	Nothing of note - -	—	Premature labour, transverse present.; version.	—	—	N.E. - -	Transverse present.	Unknown.
128	52	3,000	F.	—	Necrosis of cells of liver and kidney.		Tocanalgine given -	340	8·9	Normal - -	Drug poisoning.	
134	58	3,650	M.	—	Nothing of note - -	+	Breech labour, difficult	—	—	N.E. - -	Breech labour -	Unknown.
135	52	4,300	M.	—	Torn tentorium; cerebral hæm., K−, SR−.	—	Contracted pelvis; forceps.	—	—	N.E. - -	Contracted pelvis	Cerebral hæm.
136	46	2,450	M.	—	Signs of acute anaërosis, T+.	—	A.P.H., placenta prævia; internal version.	400	6·1	Normal - -	Placenta prævia	Acute anaërosis.
137	50	2,900	M.	—	Nothing of note, SR− -	?+	B. coli pyelitis, long first stage.	305	9·5	Normal - -	B. coli pyelitis.	
140	48	1,122	F.	—	Cerebral hæm.; signs of acute anaërosis.	—	Albuminuria; labour induced; prolapse of cord.	110	10·2	Normal - -	Albuminuria of pregnancy.	Prolapse of cord; acute anaërosis.

TABLE XIIA.—FRESH—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
142	39	1,300	M.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis.		A.P.H., placenta prævia; internal version.	170	7·6	Normal - -	Placenta prævia	Cerebral hæm.
143	46	2,000	F.	—	Hæmatoma of falx; signs of acute anaërosis, L+, S+, T+.	—	A.P.H., accidental; forceps.	260	7·6	Red infarction; retroplac hæmatoma.	Separation of N.S. placenta.	Rel. plac. insuffic.; acute anaërosis.
145	48	2,250	F.	—	Nothing of note, S+ -	—	Cord coiled four times round neck of fœtus.	240	9·3	Normal - -	Coiling of cord round fœtus.	
146	59	3,800	M.	—	Nothing of note - -	—	Prolapse of cord - -	510	7·4	Normal - -	Prolapse of cord.	
156	48	3,900	F.	—	Torn tentorium; cerebral hæm.	—	Twins; L.O.P. other twin alive; natural delivery.	—	—	N.E. - -	Excessive cranial stress.	Cerebral hæm.
157	47	2,990	F.	—	Torn tentorium; cerebral hæm., K—.		Prolapse of cord; forceps.	410	7·2	Normal - -	Prolapse of cord	Cerebral hæm.
159	43	1,710	M.	—	Hæmatoma of falx; signs of acute anaërosis, L—, T+.		A.P.H., accidental; died	—	—	N.E. - -	Separation of N.S. placenta.	Acute anaërosis.
160	55	3,400	F.	—	Nothing of note, SR+ -	—	Induction of labour for premature rupture of membranes.	470	7·2	Extensive red infarction.	Red infarction of placenta.	Rel. plac. insuffic.
163	42	2,250	M.	—	Torn tentorium; cerebral hæm.	+	Breech labour - -	340	6·6	Large avascular villi in places; "suspicions" of syphilis.	Breech labour, possible syphilis.	Cerebral hæm.
165	48	1,850	F.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis.	+	A.P.H., placenta prævia; internal version.	450	6	N.E. - -	Placenta prævia	Cerebral hæm.; acute anaërosis.

166	49	2,700	F.	—	Nothing of note - -	—	A.P.H., placenta prævia; bag; forceps; cord compressed.	—	—	N.E. - -	Placenta prævia	Compression of cord.
167	44	1,940	M.	—	Cerebral hæm.; signs of acute anaërosis.	+	A.P.H., accidental -	275	7	Red infarction	Separation of N.S. placenta; possible syphilis.	Acute anaërosis.
169	51	3,590	F.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis, SR+.	—	Prolapsed cord; forceps	380	9·3	Normal - -	Prolapse of cord	Acute anaërosis.
175	53	3,500	F.	—	Torn tentorium; cerebral hæm.	—	Contracted pelvis; pubiotomy; forceps.	520	6·7	Normal - -	Contracted pelvis	Cerebral hæm.
176	46	2,650	M.	+	Torn tentorium; cerebral hæm.; osteochondritis.	+	Former extensive perineal plastic operation; precipitate labour; perineum and vagina lacerated.	340	7·7	Typical of syphilis.	Syphilis - -	Cerebral hæm.
177	48	3,010	F.	—	Torn tentorium; cerebral hæm., K+.	—	Breech labour - -	420	7·1	Normal - -	Breech labour -	Cerebral hæm.
179	53	4,500	M.		Torn tentorium; cerebral hæm, S−.	—	Contracted pelvis; forceps.	550	8·1	- - -	Contracted pelvis	Cerebral hæm.
183	43	1,900	M.	—	Necrosis of renal epith.; nothing else of note.	—	Albuminuria - -	280	6·7	N.E. - -	Albuminuria of pregnancy.	
187	42	1,115	M.	—	Nothing of note, K+, SR+, T+.	—	A.P.H.; placenta prævia	200	5·5	Normal - -	Placenta prævia	
188	42	2,200	M.		Anencephaly, SR−, T+	—	Nil - - - -	250	8·8	Normal - -	Anencephaly.	
191	51	3,380	M.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis.		A.P.H., accidental -	280	12	Red infarction	Separation of N.S. placenta.	Cerebral hæm.
194	48	2,600	F.	—	Signs of acute anaërosis, L−.	—	Prolapse of cord - -	320	8·1	Normal - -	Prolapse of cord	Acute anaërosis.
195	42	1,750	M.	—	Cerebral hæm.; signs of acute anaërosis.	—	A.P.H., accidental -	245	7·1	N.E. - -	Separation of N.S. placenta.	Acute anaërosis.
196	48	2,600	M.	—	Torn tentorium; cerebral hæm.	—	Second twin, other fœtus alive; normal vertex labour.	280	9·3	N.E. - -	Excessive cranial stress.	Cerebral hæm.

TABLE XIIa.—FRESH—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
200	40	1,440	F.	—	Torn tentorium; cerebral hæm.	—	Breech labour	—	—	N.E.	Breech labour	Cerebral hæm.
203	50	3,350	M.	—	Much subdural cerebral hæm.; no tears of tentorium; signs of acute anaërosis, K —.	—	A.P.H.; placenta prævia	—	—	N.E.	Placenta prævia	Acute anaërosis.
204	51	2,590	M.	—	Nothing of note, S+, SR+.	+	A.P.H., accidental	270	9·5	Red infarction	Separation of N.S. placenta; possible syphilis.	
205	47	2,250	M.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis.	—	Breech labour	—	—	N.E.	Breech labour	Cerebral hæm.
206	49	2,650	M.	—	Anencephaly, T+	—.	Nil	—	—	N.E.	Anencephaly	
207	42	2,070	M.	—	Cerebral hæm.; necrosis of cells of liver and kidney.	—	Eclampsia	—	—	N.E.	Eclampsia	Fœtal toxæmia.
208	51	2,950	M.	—	Signs of acute anaërosis	—	A.P.H.; placenta prævia; internal version; prolapsed cord.	340	8·6	N.E.	Placenta prævia	Prolapse of cord and acute anaërosis.
209	49	3,400	M.	—	Torn tentorium; cerebral hæm, L —, SR —.	—	Transverse present.; internal version.	—	—	N.E.	Transverse presentation.	Cerebral hæm.
212	47	2,100	F.	—	Torn tentorium; cerebral hæm.; general œdema, K —, SR —.	—	Nil	—	—	N.E.	General œdema of fœtus.	Cerebral hæm.

216	48	2,300	M.	—	Signs of acute anaërosis -	—	Transverse present.; prolapse of cord; internal version.	—	—	N.E. - -	Transverse presentation.	Prolapse of cord and acute anaërosis.
218	47	2,350	F.	—	Signs of acute anaërosis -	—	A.P.H., accidental -	240	9·7	Red infarction	Separation of N.S. placenta.	Acute anaërosis.
220	51	4,350	M.	—	Torn tentorium and falx; cerebral hæm.; signs of acute anaërosis.	—	Brow presentation; forceps.	580	7·5	Normal - -	Brow presentation.	Cerebral hæm.
225	44	1,800	M.	—	Nothing of note, L— -	—	Slight albuminuria of pregnancy.	310	5·8	Extensive white infarction.	Extensive white infarction of placenta.	Rel. plac. insuffic.
226	36	1,110	M.	—	Torn tentorium; cerebral hæm.; ruptured liver, K+.	—	Breech labour - -	200	5·5	Normal - -	Breech labour -	Cerebral hæm., and fœtal injuries.
229	36	1,010	M.	—	Osteochondritis, L+, S+	+		330	3	N.E. - -	Syphilis - -	
231	52	3,560	M.	—	Torn tentorium; cerebral hæm.	—	Contracted pelvis; forceps; Cæsarean section.	360	9·8	Normal - -	Contracted pelvis	Cerebral hæm.
232	40	1,630	M.	—	Kidneys weighed only 1 gm. each, K—.	—	Fœtal heart heard 1 hr. before birth.	230	7	Normal - -	Under-development of fœtal kidneys.	
235	44	3,050	M.	—	Signs of acute anaërosis -	—	Contracted pelvis; labour induced; omnopon-scopolamine.	340	8·9	Normal - -	Contracted pelvis	Acute anaërosis.
236	53	3,385	F.	—	Slight tear of tentorium; cerebral hæm.	—	A.P.H., placenta prævia; prolapse of cord; forceps.	300	11·2	Normal - -	Placenta prævia	Prolapse of cord.
237	48	2,200	F.	—	Head perforated (post mortem).	—	Contracted pelvis; prolapse of cord; forceps; craniotomy.	330	6·6	Normal - -	Contracted pelvis	Prolapse of cord.
245	50	2,500	F.	—	Cerebral hæmorrhage, S+	—	Transverse presentation; internal version; prolapse of cord.	330	7·5	Normal - -	Transverse present.	Prolapse of cord.
246	42	2,170	M.	+	Osteochondritis; skin-rash, S+, K+.	+	Nil - - - -	370	5·8	Suspicions of syphilis.	Syphilis.	

TABLE XIIa.—FRESH—*continued.*

No.	Fœtus.					Mother		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
248	53	3,600	M.	—	Head perforated (post mortem), K—.	—	Contracted pelvis; forceps; craniotomy.	—	—	N.E.	Contracted pelvis.	
252	45	2,050	M.	—	Decapitated, K+	—	Contracted pelvis; transverse present.; decapitation.	—	—	N.E.	Contracted pelvis	Transverse present.
255	45	2,150	F.	—	Cerebral hæm.; signs of acute anaërosis.	—	A.P.H., placenta prævia; bag.	230	9·3	N.E.	Placenta prævia	Acute anaërosis.
258		3,250	M.	—	Head perforated (post mortem); signs of acute anaërosis.	—	Contracted pelvis; forceps; craniotomy.	—	—	N.E.	Contracted pelvis.	
259	49	2,690	M.	—	Torn tentorium; cerebral hæm., K+.	—	Contracted pelvis; labour induced; version.	—	—	N.E.	Contracted pelvis	Cerebral hæm.
261	49	2,750	F.	—	Signs of acute anaërosis		A.P.H., placenta prævia; version; septicæmia; died.	330	8·3	Normal	Placenta prævia	Acute anaërosis.
263	49	2,400	M.	—	Torn tentorium; no hæm.; signs of acute anaërosis.	—	A.P.H., placenta prævia; version.	450	5·3	Normal	Placenta prævia	Acute anaërosis
264	51	2,990	M.	—	Signs of acute anaërosis	—	Prolapse of cord	330	9	Normal	Prolapse of cord	Acute anaëros
265	47	2,650	M.	—	Torn tentorium; cerebral hæm., fœtus very anæmic, T+.	—	A.P.H., Placenta prævia; bag; forceps.	300	8·8	Normal	Placenta prævia	Cerebral hæm
268	48	2,265	F.	—	Torn tentorium; cerebral hæm., SR+.	—	Transverse present.; internal version.	—	—	Normal	Transverse present.	Cerebral hæm
269	52	3,700	F.	—	Torn tentorium; cerebral hæm.	—	A.P.H., placenta prævia; bag; forceps.	360	10·2	Normal	Placenta prævia	Cerebral hæm.
272	54	2,780	F.	—	Torn tentorium; cerebral hæm.	—	Breech labour; prolapse of cord.	450	6·1	Normal	Breech labour	Prolapse of cord and cerebral hæm.

277	43	2,120	M.	—	Signs of acute anaërosis	—	A.P.H., accidental. Fibroids; Cæsarean section.	—	—	Retroplacental hæm.; red infarction.	Separation of N.S. placenta.	Acute anaërosis.
283	55	2,800	M.	—	Torn tentorium; cerebral hæm.	—	Breech labour - -	—	—	N.E. - -	Breech labour -	Cerebral hæm.
286	45	1,750	F.	—	No histological exam. -	—	Eclampsia; died -	280	6·2	Massive red infarction.	Eclampsia.	
288	47	3,890	M.	—	Head not examined, K—	—	Prolapse of cord; forceps.	—	—	N.E. - -	Prolapse of cord.	
293	47	3,050	M.	—	Torn tentorium; cerebral hæm.	—	A.P.H., accidental -	335	9·1	Red infarct. -	Separation of N.S. placenta.	Cerebral hæm.
294	44	2,170	F.	—	Subdural cerebral hæm.; no tear of tentorium, S+.	—	A.P.H., accidental -	340	6·3	Normal - -	Separation of N.S. placenta.	
295	44	1,345	M.	—	Nothing of note, L+, S+, K+, T+.	—	A.P.H., placenta prævia; bag; internal version.	260	5·1	Normal - -	Placenta prævia.	
296	40	1,200	M.	—	Nothing of note - -	—	A.P.H., placenta prævia; bag; breech labour.	220	5·4	N.E. - -	Placenta prævia.	
298	46	2,894	F.	—	Nothing of note, T— -	—	Normal labour; vertex	—	—	N.E. - -	Unknown.	
301	51	3,540	M.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis, SR—.	—	Transverse present.; prolapse of cord, version.	470	7·5	Normal - -	Transverse present.	Prolapse of cord; cerebral hæm.; acute anaërosis.
302	51	3,450	M.	—	Torn tentorium; no hæm., K—, T+.	—	A.P.H., placenta prævia; internal version.	460	7·5	Normal - -	Placenta prævia.	
303	49	3,275	M.	M.	Torn tentorium; cerebral hæm, K—.	—	Contracted pelvis; prolapse of cord; version.	—	—	N.E. - -	Contracted pelvis	Prolapse of cord and cerebral hæm.
305	48	2,580	F.	—	Torn tentorium and falx; cerebral hæm., K—, T—.	—	Brow presentation; forceps; version.	—	—	N.E. - -	Brow presentation.	Cerebral hæm.
311	53	4,010	M.	—	Torn tentorium; cerebral hæm.	—	Persistent occipito-posterior presentation; forceps.	630	6·3	Normal - -	Long labour; O.P	Cerebral hæm.
312	50	3,270	F.	—	Torn tentorium; cerebral hæm.	—	Natural delivery; vertex; long labour.	405	8	N.E. - -	Long labour -	Cerebral hæm.

TABLE XIIA.—FRESH—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
314	45	3,200	F.	—	Torn tentorium; cerebral hæm.	—	Breech labour	380	8·5	Normal	Breech labour	Cerebral hæm.
315	48	3,700	F.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis, K—.	—	Prolapsed cord; forceps	510	7·2	Normal	Prolapsed cord	Cerebral hæm; acute anaërosis
317	37	1,275	M.	—	Torn tentorium; no hæm.	—	A.P.H., accidental	165	7·7	Normal	Separation of N.S. placenta.	
325	52	3,010	M.	—	Cerebral hæmorrhage; no tear of tentorium.	—	Born before arrival of midwife; all attendants intoxicated.	—	—	N.E.	- - -	Cerebral hæm.
326	44	2,960	M.	—	Hæmatoma of falx; signs of acute anaërosis.	—	A.P.H., placenta prævia; bag.	—	—	N.E.	Placenta prævia	Acute anaërosis.
331	51	4,250	M.	—	Perforation of head; B. coli cultivated from heart-blood; signs of acute anaërosis.	—	Obstructed labour; craniotomy; rupture of uterus; death.	390	10·8	N.E.	Contracted pelvis	
334	45	2,058	F.	—	One of twins; nothing of note, T—.	—	Breech labour; twins	—	—	N.E.	Breech labour.	
337	42	1,620	F.	—	Torn tentorium; cerebral hæm., K—.	—	Transverse presentation; spontaneous evolution.	—	—	N.E.	Transverse presentation.	Cerebral hæm.
338	46	2,690	M.	—	Torn tentorium; no hæm.	—	A.P.H., placenta prævia	320	8·4	Red infarction	Placenta prævia.	
339	51	4,380	M.	—	Torn tentorium; cerebral hæm; signs of acute anaërosis, SR+, T+.	—	Brow presentation; version.	—	—	N.E.	Brow presentation.	Cerebral hæm
340	47	3,690	M.	—	Torn tentorium; cerebral hæm, T+.	—	Forceps; difficult delivery.	—	—	N.E.	Long labour; forceps.	Cerebral hæm.
341	45	3,050	M.	—	Torn tentorium; cerebral hæm.	—	Prolapsed cord; twins	—	—	N.E.	Prolapsed cord	Cerebral hæm.

343	41	2,159	F.	—	Torn tentorium; cerebral hæm., T+.	?	A.P.H., placenta prævia; internal version.	272	7·9	N.E.	Placenta prævia	Cerebral hæm.
344	38	2,170	F.	—	Torn tentorium; cerebral hæm, T+.	—	A.P.H., placenta prævia; internal version.	217	9·9	Normal	Placenta prævia	Cerebral hæm.
346	54	3,600	M.	—	No cerebral hæm.; nothing of note, L+	—	Forceps delivery; vertex.	—	—	N.E.	Contracted pelvis	
347	47	2,170	M.	—	Cerebral hæm.; signs of acute anaërosis.	—	Natural delivery; vertex.	—	—	N.E.	Unknown	Acute anaërosis
349	54	3,370	M.	—	Head perforated, T+	—	Contracted pelvis; induction of labour; pituitrin; tonic contraction of uterus; forceps; craniotomy.	370	9·1	Normal	Contracted pelvis	Tonic uterine contraction.
350	47	3,220	F.	—	Torn tentorium; cerebral hæm, S+	—	Contracted pelvis; forceps.	410	7·8	Normal	Contracted pelvis	Cerebral hæm
352	46	2,060	M.	—	The second of twins; no cerebral hæm.	—	Twins; born before arrival of attendant.	—	—	N.E.	Unknown.	
353	51	3,710	F	—	Torn tentorium; cerebral hæm.	—	A.P.H., placenta prævia; bag.	390	9·5	Normal	Placenta prævia	Cerebral hæm.
355	49	2,780	M.	—	Torn tentorium; cerebral hæm., S+, SR+, T+.	—	Forceps; long labour; inertia; manual rotation of head to O.A.	—	—	N.E.	Long labour	Cerebral hæm.
356	56	3,550	F.	—	Torn tentorium and falx; cerebral hæm.; signs of acute anaërosis, L+.	—	Breech labour, delay with head; forceps.	—	—	N.E.	Breech labour	Cerebral hæm.
358	50	3,070	M.	—	Torn tentorium; cerebral hæm.	—	Forceps; prolonged 2nd stage; vertex; O.A.	—	—	N.E.	Long labour	Cerebral hæm.
360	46	2,160	M.	—	Torn tentorium; cerebral hæm	?	A.P.H., placenta prævia; bag; prolapse of cord.	410	5·2	Normal	Placenta prævia	Prolapse of cord and cerebral hæm.
361	47	2,570	M.	—	Torn tentorium; cerebral hæm.	—	Twins; forceps; vertex; long 2nd stage.	—	—	N.E.	Long labour	Cerebral hæm.
363	52	3,050	M.	—	Torn tentorium; cerebral hæm.	—	Breech labour	—	—	N.E.	Breech labour	Cerebral hæm.
364	45	2,590	F.	—	Torn tentorium; cerebral hæm.	—	Vertex; natural delivery	—	—	N.E.	Excessive cranial stress.	Cerebral hæm.

TABLE XIIa.—FRESH—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
365	36	1,100	M.	—	General œdema, S+, SR+, T−.	—	Natural delivery; repeated premature labours.	690	1·8	Large, dense, avascular villi; "Suspicions" of syphilis.	General œdema of fœtus.	
366		2,450	M.	—	Cerebral hæm.; signs of acute anaërosis.	—	A.P.H., accidental; albuminuria; Cæsarean section.	—	—	N.E.	Separation of N.S. placenta.	Acute anaërosis.
368		2,460	M.	—	Cerebral hæm.; signs of acute anaërosis, K+.	—	A.P.H., accidental; pituitrin; nat. delivery.	370	6·4	N.E.	Separation of N.S. placenta.	Acute anaërosis.
369	49	3,150	F.	—	Torn tentorium; cerebral hæm.	—	Precipitate labour; uncompensated mitral stenosis.	340	9·2	Normal	Precipitate labour.	Cerebral hæm.
372	42	2,075	F.	—	Cerebral hæm; no tear of tentorium.	—	Albuminuria; normal labour.	200	10·3	Several white infarcts.	Albuminuria of pregnancy.	
373	48	1,870	M.	—	Cerebral hæm.; signs of acute anaërosis, SR+.	—	A.P.H., placenta prævia; natural delivery.	170	11	Normal	Placenta prævia	Acute anaërosis.
374	50	3,690	F.	—	Torn tentorium; cerebral hæm, K+, SR+.	—	A.P.H., placenta prævia; transverse presentation; version.	410	9	N.E.	Placenta prævia	Cerebral hæm.
379	47	2,767	F.	—	Nothing of note	—	A.P.H., placenta prævia.	300	9·2	Red infarction	Placenta prævia.	
381	52	3,070	F.	—	Cerebral hæm.; signs of acute anaërosis.	—	Prolapsed cord; vertex present.	—	—	N.E.	Prolapsed cord	Acute anaërosis.
383	39	1,160	M.	—	Nothing of note, S−, SR+.		A.P.H., placenta prævia; internal version.	240	4·8	Normal	Placenta prævia	
386	52	3,120	F.	—	Torn tentorium; cerebral hæm, K+.	—	Contracted pelvis; prolapsed cord; internal version.	—	—	N.E.	Contracted pelvis	Prolapsed cord and cerebral hæm.

389	47	3,070	F.	—	Hæmatoma of falx; signs of acute anaërosis.		Albuminuria of pregnancy.	450	6·8	Hyaline degen. excessive, villi "suspicious" of syphilis.	Albuminuria of pregnancy.	Acute anaërosis.
390	41	1,470	M.	—	Torn tentorium; cerebral hæm.; one of binovular twins, the other was a fœtus papyraceus.	—	A.P.H., accidental; natural delivery; vertex.	225	6·5	Normal - -	Separation of N.S. placenta.	Cerebral hæm.
393	36	1,040	M.	—	Liver ruptured; much blood in peritoneal cavity.	—	Hydramnion; transverse presentation; breech labour.	—	—	N.E. - -	Transverse presentation.	Fœtal injury.
395	37	2,360	M.	—	General œdema, K —, T —	—	A.P.H., placenta prævia; natural delivery.	—	—	N.E. - -	Placenta prævia; general œdema of fœtus.	
397	54	3,215	M.	—	Torn tentorium; cerebral hæm.	—	Breech labour - -	—	—	N.E. - -	Breech labour -	Cerebral hæm.
398	50	2,670	M.	—	Torn tentorium; cerebral hæm; signs of acute anaërosis	—	Albuminuria, induction of labour; vertex.	—	—	N.E. - -	Albuminuria of pregnancy.	Cerebral hæm.
400	52	3,010	M.	—	Torn tentorium; cerebral hæm., K +.	—	Vertex; natural delivery	—	—	N.E. - -	Excessive cranial stress.	Cerebral hæm.
401	53	3,025	M.	—	Torn tentorium; cerebral hæm	—	Breech labour - -	520	5·8	Red necrosis -	Breech labour -	Cerebral hæm.
404*a*	54	3,100	F.	—	Torn tentorium; no hæm.	—	Albuminuria; twins; placenta prævia; int. version; died.	290	10·6	N.E. - -	Albuminuria of pregnancy and placenta prævia.	Cerebral hæm.
b	51	2,820	F.	—	Torn tentorium; cerebral hæm., S —, SR +.	—		280	10	N.E. - -		
405	36	1,150	M.	—	Premature; nothing of note.		Natural labour - -	283	4	N.E. - -	Prematurity.	
406	51	2,670	M.	—	Torn tentorium; cerebral hæm., L +, S +, T +.	+	Spontaneous labour; L.O.P.	440	6	Normal - -	Excessive cranial stress; probable syphilis.	Cerebral hæm.
409	51	2,490	F.	—	A few signs of acute anaërosis; no cerebral hæm.		Contracted pelvis; induction; forceps; cord coiled round neck and trunk of fœtus, and relatively short.	—	—	N.E. - -	Contracted pelvis	Constriction of cord.

TABLE XIIa.—FRESH—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Changes, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
410	48	3,150	M.	—	Head perforated - -	—	Contracted pelvis; craniotomy.	—	—	N.E. - -	Contracted pelvis	
411	51	3,250	M.	—	Nothing of note - -	?	Prolapse of cord; uncompensated mitral disease; vertex.	—	—	N.E. - -	Prolapse of cord.	
414	49	3,290	M.	—	Torn tentorium; cerebral hæm.	—	Face presentation; long labour.	—	—	N.E. - -	Face presentation.	Cerebral hæm.
415	52	3,070	F.	—	Torn tentorium; cerebral hæm.	—	Forceps; vertex present.	—	—	N.E. - -	Long labour -	Cerebral hæm.
417	55	3,230	M.	—	Head perforated - -	—	Contracted pelvis; craniotomy.	481	6·7	N.E. - -	Contracted pelvis.	
418	50	3,203	F.	—	Torn tentorium; cerebral hæm.; signs of acute anaërosis.	—	Contracted pelvis; forceps.	—	—	N.E. - -	Contracted pelvis	Cerebral hæm.; acute anaërosis.
419	53	3,430	M.	—	Fœtus long and poorly nourished, K+, T−.	—	Postmaturity of fœtus; pregnancy prolonged by five weeks.	481	7·1	Hyaline degeneration excessive; areas of white infarction.	Postmaturity -	Relative placental insuffic.
420	50	2,750	F.	—	Torn tentorium; cerebral hæm.; extreme moulding of head.	—	Contracted pelvis; forceps.	—	—	N.E. - -	Contracted pelvis	Cerebral hæm.
421	52	3,061	M.	—	Torn tentorium; cerebral hæm., S−, SR−.	—	Natural delivery: vertex; O.A.	—	—		Excessive cranial stress; ? cause.	Cerebral hæm.
422	55	4,536	—	—	Torn tentorium; cerebral hæm., L+, K−.	—	Forceps; occipito-posterior.	—	—		Contracted pelvis	Cerebral hæm.

TABLE XIIB.—TABLE OF CASES: MACERATED FŒTUSES: 133.

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Findings, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause	Consequent Factors.
4		1,940	F.	+	Osteochondritis, S+, T−	+	Nil	360	5·4	N.E.	Syphilis.	
5		3,628	F.	−	Hydrocephalus, spina bifida, double talipes equinus, S+, K+, T−	−	Nil	410	8·8	Normal	Multiple fœtal deformities.	
7		?	F.	−	Nothing of note	−	Habitual fœtal death	560		Multiple interstitial hæmorrhages.	Intraplacental hæm.	Relative placent. insuffic.
11	54	2,010	M.	−	Nothing of note	−	Fell on abdomen a week before labour.	270	7·4	Retroplacental hæmatoma and red infarction.	Retroplac. hæmatoma.	Relative placent. insuffic.
12	39	1,430	M.	−	Osteochondritis	+	Signs of congenital syphilis; transverse present., prolapse of cord.	280	5	Typical of syphilis	Syphilis.	
14	38	1,990	F.	+	Osteochondritis, L+, S+, K+ S.R.+	+	Nil	360	5·5	Typical of syphilis	Syphilis.	
18	46	3,451	M.	−	Nothing of note, K−	−	Nil	669	5·1	N.E.	Unknown.	
21	30	1,090	M.	+	Osteochondritis, L+, S+, K−.	+	Nil	330	3·3	Typical of syphilis	Syphilis.	
22	42	1,920	F.		Nothing of note	+	A.P.H., placenta prævia.	—	—	Normal	Placenta prævia.	
25	39	1,134	F.	−	Nothing of note	−	Nil; breech labour	150	7·5	N.E.	Unknown.	
26	42	1,728	F.	−	Nothing of note	−	A.P.H., accidental	260	6·6	Extensive red infarction.	Separation of N.S. placenta.	
29		1,470	F.	+	Osteochonditis, L+, S+, K+.	+	Nil	280	5·2	Suspicions of syphilis	Syphilis.	
30		4,149	M.	−	Nothing of note, L−, S−, K−, SR−.	−	Nil	320	12·9	Excessive hyaline degen. of villi.	Small placenta.	Relative placent. insuffic.

TABLE XIIB.—MACERATED—*continued.*

No	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Findings, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
31		1,474	F.	+	Osteochondritis, L+, S+, K+.	+	Condylomata on vulva.	470	3·1	N.E.	Syphilis.	
32		3,032	M.	—	R. hypogastric and umbilical arteries absent, S—.	—	Nil	270	11·2	Moderate hyaline degen. of villi.	Absence of umbil. vessel and small placenta.	Relative placent. insuffic.
34		2,621	M.	—	Nothing of note, S+, S.R.—.	—	Anæmia; R.B.C., 3,000,000; Hbgm. 60 per cent.	240	10·9	Moderate hyaline degen. of villi.	Small placenta	Relative placent. insuffic.
38	55	2,494	M.	—	Nothing of note, T—	—	Albuminuria; A.P.H., accidental.	280	8·9	Villi large and dense; "suspicions" of syphilis.	Albuminuria of pregnancy; accidental hæm.	
40	35	800	F.	+	Osteochondritis, S+	+	Nil	260	3	Typical of syphilis	Syphilis.	
41	49	2,704	M.	+	Osteochondritis, S+, T+	+	Nil	370	7·3	Much irregular white infarction.	Syphilis.	
44	42	970	M.	—	Left hypogastric and umbilical arteries absent; talipes; spina bifida.	—	A.P.H. accidental	176	5·5	Excessive white and red infarcts. and hyaline degen. of villi.	Separation of N.S. placenta.	
49	44	1,771	F.	—	Nothing of note, K—, SR—.	—	Eclampsia	—	—	N.E.	Eclampsia.	
53	36	625	M.	+	Osteochondritis, L+, S+, K+.	+	Nil	160	3·8	Typical of syphilis	Syphilis.	
60	51	2,850	M.	—	Left hypogastric and umbilical arteries absent; hydrocephalus, K—, SR—.	—	Nil	385	7·4	Hyaline degen. and avascularity of villi excessive.	Absence of umbil. art.	Relative placent. insuffic.

61	42	1,000	F.	+	Osteochondritis, L+, S+, K+, SR+.	—	Nil	320	3·1	Typical of syphilis	Syphilis.
62	45	2,500	F.	+	Osteochondritis, L+, S+, T+.	+	Nil	320	7·8	Typical of syphilis	Syphilis.
65	40	1,950	F.	—	Osteochondritis, S+	+	Nil	650	3	Typical of syphilis	Syphilis.
68	47	1,820	M.	—	Nothing of note, S+	+	Nil	560	3·2	Normal	Unknown; possible syphilis.
79	40	1,750	F.	+	Osteochondritis, S+	+	Breech labour	250	7	Typical of syphilis	Syphilis.
80	42	1,190	F.	—	Nothing of note, S—	+	Albuminuria of pregnancy, induction of labour.	—	—	N.E.	Albuminuria of pregnancy.
84	50	3,300	M.	+	Osteochondritis, L+, S+, K+.	+	Hydramnion	—	—	N.E.	Syphilis.
102	39	1,050	F.	+	Osteochondritis, SR+	+	Albuminuria of pregnancy.	170	6·1	Normal	Syphilis.
104	36	930	M.	—	Nothing of note	—	Twins; other fœtus living; hydramnion.	—	—	N.E.	Hydramnion, uniovular twins.
105	40	1,510	M.	—	Nothing of note, L+, S+, K—, SR+, T—.	—	Albuminuria of pregnancy.	—	—	N.E.	Albuminuria of pregnancy.
109	37	1,100	F.	+	Osteochondritis, L+, S+, K+.	—	Nil	225	4·8	N.E.	Syphilis.
110	40	1,170	M.	—	Nothing of note, S+, K+.	—	Albuminuria of pregnancy.	220	5·3	Red infarction	Albuminuria of pregnancy.
115	36	1,040	M.	—	No evidence of tubercle in fœtus.	—	Advanced pulmonary tuberculosis.	—	—	N.E.	? Pulmonary tuberculosis in mother.
118	36	1,250	F.	+	Osteochondritis, L+, S+, K+, SR+.	+	Nil	220	5·6	Typical of syphilis	Syphilis.
122	46	2,050	F.	—	Nothing of note, L—, K—, SR—.	—	Nil	—	—	N.E.	Unknown.
124	48	2,550	F.	—	No osteochondritis, S—	+	Nil	260	9·8	Typical of syphilis	Syphilis.
127	50	2,891	F.	—	Nothing of note	—	Albuminuria of pregnancy.	300	9·6	Many white infarcts	Albuminuria of pregnancy.
129	46	1,550	M.	+	Osteochondritis, S+	+	Nil	220	7	Suspicions of syphilis	Syphilis.
132	43	1,900	M.	+	Osteochondritis, S+	+	Nil	190	10	Typical of Syphilis	Syphilis.

TABLE XIIB.—MACERATED—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Findings, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
133	45	2,500	F.	—	Nothing of note	—	A.P.H., accidental	300	8·3	Large red infarct; many large avascular villi; "suspicions" of syphilis.	Separation of N.S. placenta.	
141	35	750	F.	+	Osteochondritis, S+	+	Placenta prævia	185	4	Normal	Syphilis.	
148	39	1,040	F.	+	Osteochondritis, S+, T+, K−.	+	Nil	152	6·7	Suspicions of syphilis	Syphilis.	
149	37	1,300	F.	+	Osteochondritis, T+	+	A.P.H., placenta prævia.	200	6·5	N.E.	Syphilis.	
150	47	2,950	M.	+	Nothing of note, S−, K−.	—	Nil	310	9·5	N.E.	Unknown.	
152	40	1,300	F.	—	Nothing of note, L+, S+, K+.	—	Hydramnion	290	4·4	Normal	Unknown.	
164	36	1,040	F.	—	Nothing of note, L+	+	Nil	—	—	N.E.	Unknown; possible syphilis.	
168	51	2,550	M.	—	Pure growth of coliform bacillus from heart-blood, S+, SR−, T+.	—	Nil	460	5·5	Normal	? B. coli infection of fœtus.	
170	52	3,590	M.	—	Signs of acute anaërosis, K−, T+.	—	A.P.H., placenta prævia.	410	8·7	Hyaline degen. excessive.	Placenta prævia.	Acute anaërosis
180	53	3,800	F.	—	Signs of acute anaërosis; very early maceration.	—	A.P.H., placenta prævia; version.	350	10·8	N.E.	Placenta prævia.	Acute anaërosis
181	46	2,670	F.	+	Ostrochondritis, K+	+	Nil	350	7·6	Typical of syphilis	Syphilis.	
184	51	3,200	F.	—	Nothing of note	—	Labour lasted 72 hours.	290	11	N.E.	Small placenta	Relative placent. insuffic.
185	43	1,690	M.	—	v. slight maceration	+	Short labour; nil	155	11	Normal	Small placenta.	

186	47	1,200	F.	+	No osteochondritis, L+, S+, SR+, K+, signs of acute anaërosis.	+	Nil	—	—	N.E.	Syphilis.	
189	40	1,180	M.	—	Nothing of note, SR+	—	Nil	—	—	N.E.	Unknown.	
190	41	2,660	M.	—	Nothing of note, L+, S+	—	Albuminuria of pregnancy.	700	3·8	Very large and pale; two small intraplacental hæms.; villi very large and avascular, but stroma loose.	Albuminuria of pregnancy.	
192	45	2,500	M.	—	Acute anaërosis	—	A.P.H., concealed, accidental; natural delivery.	290	8·6	Red infarction	Separation of N.S. placenta.	Acute anaërosis
193	40	2,250	F.	+	Osteochondritis, S+	+	Nil	410	5·4	Typical of syphilis	Syphilis.	
197	49	3,150	M.	—	Acute anaërosis, S+, K—	—	No external A.P.H.	400	7·8	Large retroplacental hæmatoma.	Retroplac. hæmatoma.	Acute anaërosis
198	42	1,640	M.	—	Nothing of note, S—, T—	—	Albuminuria of pregnancy.	180	9·1	A few groups of large villi; "suspicions of syphilis."	Albuminuria of pregnancy.	
201	49	2,400	F.	—	Coliform bacilli cultivated from heart-blood, K+.	—	First stage lasted 96 hours with ruptured membranes.	360	6·5	N.E.	B. coli. infection	
210	41	1,120	M.	—	Nothing of note, L—	—	Nil	290	3·8	Many enlarged dense villi, as in typical syphilitic placenta.	probable syphilis	
211	41	2,700	F.	—	Nothing of note, S+	+	Nil	340	7·9	Normal	Unknown; possible syphilis.	
215	48	2,580	F.	—	Nothing of note, SR—	—	Nil	330	7·8	Normal	Unknown.	
217	41	1,070	F.	—	Nothing of note, S—	—	Albuminuria of pregnancy.	120	8·9	Large intraplacental hæmorrhage.	Albuminuria of pregnancy.	Relative placent. insuffic.
219	36	1,550	M.	—	Nothing of note	—	Nil	—	—	N.E.	Unknown.	
221	48	2,200	F.	—	Nothing of note, SR—	—	Nil	350	6·2	Normal	Unknown.	
222	39	1,800	M.	+	Osteochondritis, S+	+	Nil	—	—	N.E.	Syphilis.	
224	45	1,570	F.	—	Nil of note, L—	—	Triplets, one alive	—	—	N.E.	Unknown.	

TABLE XIIB.—MACERATED—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Findings, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
227	33	590	M.	+	Double talipes equinus, osteochondritis, S+, SR+.	+	Nil - - - -	210	2·8	Suspicions of syphilis.	Syphilis.	
228	47	2,500	M.	—	Signs of acute anaërosis, S—.	—	No external A.P.H. -	280	8·9	Intraplacental hæm.; retroplacental hæmatoma; red infarction.	Retro- and intra-placental hæmorrhage.	Relative placent. insuffic.
230	52	4,050	F.	—	Very large fœtus; had been eviscerated, L—, SR—.	—	One month postmature; obstructed labour; evisceration.	430	9·4	Villi normal; a few infarcts.	Postmaturity.	
234	42	2,200	M.	—	L+, S+, K+, SR+ -	+	Four former deadbirths.	—	—	N.E. - - -	Probable syphilis.	
238	55	4,880	F.	—	Nothing of note; very large fœtus, K—, SR—.	—	Hydramnion - -	510	9·5	Hyaline degen. excessive.	Unknown.	
239	51	3,200	M.	—	Nothing of note - -	—	Nil - - - -	—	—	N.E. - - -	Unknown.	
243	40	1,365	M.	+	Osteochondritis, S+ -	+	Nil - - - -	—	—	Suspicions of syphilis.	Syphilis.	
247	36	2,070	M.	—	Nothing of note, L+, S+, T+.	—	Chronic renal disease, severe.	—	—	N.E. - - -	Chronic renal disease.	
251	39	1,750	M.	—	Nothing of note, S—, K—.	—	Pulmonary tuberculosis, advanced.	180	9·7	No tubercle bacilli; villi normal.	? Pulmonary tuberculosis in mother.	
257	36	885	M.	—	Nothing of note, SR— -	—	A.P.H., chronic renal disease.	—	—	N.E. - - -	Chronic renal disease.	
262	48	1,800	M.	—	Nothing of note, T— -	—	A.P.H., accidental -	—	—	N.E. - - -	Separation of N.S. placenta.	

267	51	2,700	M.	—	Nothing of note, K+	—	Transverse present.; prolapse of cord.	—	—		Transverse presentation.	Prolapse of cord.
270	42	1,040	M.	—	Nothing of note, L−, S−	—	No albuminuria	—	—	N.E.	Unknown.	
271	51	2,410	F.	—	Very slight maceration, L+.	—	A.P.H. placenta prævia.	—	—	N.E.	Placenta prævia.	
273	51	4,590	M.	—	Very large fœtus, K−, SR−.	—	Nil	340	13·5	Hyaline degen. excessive.	Small placenta.	
275	43	2,100	F.	—	Anencephaly	—	Nil	312	6·7	Normal	Anencephaly.	
278	47	2,950	M.	—	Nothing of note, K−, T+.	—	Former syphilis, treated.	250	11·8	Normal	Small placenta.	
279	47	2,508	M.	—	Nothing of note	—	Albuminuria; induction of labour.	—	—	N.E.	Albuminuria of pregnancy.	
280	39	780	M.	+	Nothing of note, T−	—	A.P.H. accidental	170	4·5	Large intraplacental hæm.	Separation of N.S. placenta.; intraplacental hæm.	
281	38	980	F.	—	Nothing of note, T+		Acute pneumonia; died; natural labour.	260	3·7	Hyaline degen. excessive.	Pneumonia in mother.	
282	42	2,110	F.	—	Left K and SR absent: rt K extremely small, T+.	—	A.P.H., accidental	380	5·5	Much red infarction	Separation of N.S. placenta.	
287	42	1,350	M.	—	Nothing of note	—	Slight albuminuria	290	4·6	Retroplacental hæmatoma; red infarct.	Retroplacental hæmatoma.	
290	54	2,585	M.	—	L+, S+, K+, SR+, signs of acute anaërosis.	+	Nil	510	5	Normal	Probable syphilis.	
291	37	1,290	F.	—	Nothing of note	—	A.P.H., accidental	390	3·3	Red infarction	Separation of N.S. placenta.	
292	45	2,160	F.	—	Nothing of note, K−	—	Syphilis treated before and during pregnancy.	400	5·5	Villi normal	Probable syphilis.	
297	32	759	M.	—	Nothing of note, S−, T−.	—	Chronic renal disease; labour induced.	135	5·6	Villi normal; many white infarcts.	Chronic renal disease.	
308	46	2,700	M.	—	Neck constricted by umbilical cord, T+.	—	Transverse present.; cord coiled tightly round neck of fœtus.	360	7·5	Normal; cord 62 cms. long.	Transverse present.	

TABLE XIIb.—MACERATED *continued*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Age (Weeks)	Wt.	Sex.	Sp	Post mortem Findings, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
309	50	3,466	F.	—	Nothing of note, L+	—	Chronic renal disease; labour induced.	450	7·6	Villi "suspicious" of syphilis.	Chronic renal disease.	
313	45	2,890	M.	—	No osteochondritis, L+, S+.	+	Gave birth to a syphilitic fœtus one year ago. (*See* No. 41.)	385	7·5	Normal	Syphilis.	
319	47	2,120	M.	—	Nothing of note, L+, K+, SR+.	—	Nil, O albuminuria	320	6·6	Normal	**Unknown.**	
321	37	1,050	M.	—	Nothing of note, S	—	Nil	—	—	N.E.	Unknown.	
322	47	2,350	M.	—	Nothing of note	—	Nil	220	10·7	Extensive white infarction.	Small placenta	Relative placent. insuffic.
323	43	2,125	M.	—	Nothing of note, L—, S—, K—.	—	Nil	170	12·5	N.E.	Small placenta	Relative placent. insuffic.
328	46	1,125	F.	+	Talipes equinus, osteochondritis, L+, S+, K+, SR+.	+	Nil	335	3·3	Normal	Syphilis.	
332	41	2,000	F.	+	Osteochondritis, L+, S+.	+	Nil	520	3·8	Typical of syphilis	Syphilis.	
335	43	2,050	F.	—	Nothing of note, L+	—	A.P.H., accidental	470	4·3	Extensive red infarction, enlarged dense villi, as in typical syphilitic placenta.	Separation of N.S. placenta; probable syphilis.	
336	35	1,010	F.	—	Nothing of note, K—	—	Nil, O albuminuria	—	—	N.E.	Unknown.	
342	45	2,510	M.	+	Osteochondritis, S+	—	Nil	225	11·1	N.E.	Syphilis.	
348	35	1,080	F.	—	Left hypogastric and umbilical arteries absent	—	Nil	160	6·7	Hyaline change excessive.	Absence of umbilical artery.	Relative placental insufficiency.

357	41	2,070	M.	—	Nothing of note, L—, S—.	—	Nil	230	8·9	Retroplacental clot; villi "suspicious" of syphilis.	Retroplacental haematoma.	
359	39	2,100	M.	—	One of twins; nothing of note, L—, S—, T—.	—	Albuminuria of pregnancy; twins.	280	8	N.E.	Albuminuria of pregnancy.	
362	35	3,770	M.	—	Nothing of note, S+, K+, SR+.	—	Albuminuria of pregnancy.	—	—	N.E.	Albuminuria of pregnancy.	
367		1,270	M.	—	Nothing of note, T+	—	A.P.H., accidental; albuminuria; died.	160	7·9	Normal	Separation of N.S. placenta.	
370	44	2,050	M.	—	Nothing of note, L+	+	Albuminuria of pregnancy.	230	8·9	N.E.	Albuminuria of pregnancy.	
371	51	3,760	F.	—	Nothing of note	—	Pneumonia; forceps	280	13·3	Normal	Small placenta.	
376	45	1,350	M.	—	Nothing of note	—	Nil; O albuminuria	320	24·2	Normal	Unknown.	
377	47	2,020	M.	+	Osteochondritis, L+, S+, K+, SR+.	+	Nil	400	5	Typical of syphilis	Syphilis.	
382	45	1,590	M.	—	Nothing of note, L+, K+, T+.	—	Albuminuria of pregnancy.	—	—	N.E.	Albuminuria of pregnancy.	
384	48	2,650	M.		Nothing of note, S+, SR+.	—	Nil	—	—	N.E.	Unknown.	
385	37	1,300	F.	—	One of twins; nothing of note.	—	Twin, other living	—	—	N.E.	Unknown.	
387	47	2,340	M.	—	Nothing of note	—	Nil	190	12·3	Normal	Small placenta.	
388	48	2,110	M.	—	Nothing of note, K—	—	Nil	—	—	N.E.	Unknown.	
391	40	1,240	F.	+	Osteochondritis, L+, S+, K+, SR+.	+	Nil	—	—	N.E.	Syphilis.	
392	52	2,500	F.	—	Umbil cord coiled tightly round body, forming grooves	—	Face present; cord wound tightly round neck and trunk of fœtus.	—	—	N.E.	Coiling of cord.	
394	38	1,138	M.	—	Nothing of note, L—, SR+.	—	Nil, O albuminuria	160	7·1	Normal	Unknown.	
396	44	2,420	M.	+	Osteochondritis, S+, T+	+	Nil	—	—	N.E.	Syphilis.	
399	36	1,220	M.	+	Osteochondritis, S+, K—.	+	Nil	—	—	N.E.	Syphilis.	
403A	47	2,420	F.		Twins, nothing of note, S+, SR+.	?	Albuminuria of pregnancy; induction of labour.	—	—	N.E.	Albuminuria of pregnancy.	
403B	51	2,500	F.									

TABLE XIIB.—MACERATED—*continued.*

No.	Fœtus.					Mother.		Placenta.			Causation of Death.	
	Length.	Wt.	Sex.	Sp.	Post mortem Findings, Deformities, &c.	W.R.	Obstetrical and other Complications.	Wt.	Ratio	Macro. and Microscop. Changes.	Primary Cause.	Consequent Factors.
407	44	2,480	M.	—	Nothing of note, SR+	?	Neglected transverse present.; died.	340	7·2	Normal	Unknown.	
412	52	3,120	M.	—	One hypogastric and umbilical artery absent, K—.	—	A.P.H., accidental	430	7·2	Red infarction	Separation of N.S. placenta.	
413	39	1,100	M.	+	Osteochondritis, S+, T+.	+	Nil	280	3·9	Typical of syphilis	Syphilis.	
416	41	1,842	M.	+	Osteochondritis, L+, S+.	+	A.P.H., accidental	490	3·7	Typical of syphilis, also retroplac. hæmatoma and red infarction.	Syphilis or separation of N.S. placenta.	

TABLE XIII.—RATIO LIST.

Fresh Fœtuses.

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
13	7·4	74	364	37	544	628	129	4,082	55
17	?	44	292	26	652	445	131	1,960	45
19	7·8	48	500	20	444	?	95	2,000	42
27	?	63	552	28	370	484	156	3,146	50
28	6·1	41	330	22	412	1,100	162	1,650	40
33	?	61	397	26	374	454	138	3,175	52
35	9	—	1,133	25	441	528	—	3,968	—
36	?	—	680	27	531	531	117	1,700	—
42	4·7	39	418	14	371	304	119	1,672	43
45	?	73	391	18	329	478	152	4,110	56
46	?	—	—	?	?	—	—	—	—
47	?	69	329	26	?	364	89	3,458	50
48	2·2	32	812	22	289	433	124	1,300	41
51	?	58	162	21	347	400	108	2,600	45
55	5·3	29	275	25	550	244	75	1,100	38
57	?	48	533	25	300	368	57	2,400	50
64	?	52	242	21	242	311	92	2,300	44
75	9·4	54	156	19	313	940	67	2,820	52
76	?	42	291	19	583	292	100	1,750	42
82	11·5	58	230	19	428	600	128	3,000	52
87	5·5	64	2,226	20	148	607	219	3,340	52
103	7·4	61	327	19	490	181	89	2,450	40
107	?	63	225	20	274	788	107	3,150	50
108	5·9	64	237	20	442	349	123	3,320	52
111	8·3	?	467	18	316	246	107	3,320	—
113	4·9	29	208	18	416	248	90	1,040	36
116	?	46	178	22	512	602	133	2,050	45
123	?	28	214	21	404	361	94	1,010	36
128	8·8	58	400	19	428	416	154	3,000	52
134	?	63	365	18	253	486	149	3,650	58
135	?	83	286	22	374	782	191	4,300	52
136	6·1	53	175	20	377	306	131	2,450	46
137	9·5	58	580	29	446	645	105	2,900	50
140	10·2	23	—	20	320	321	75	1,122	48
142	7·6	33	309	24	394	302	130	1,300	39
143	7·6	44	117	12	190	351	108	2,000	46
145	9·3	47	562	17	187	511	132	2,250	48
146	7·4	64	223	22	316	507	141	3,800	59
156	?	81	390	21	433	488	153	3,900	48
157	7·2	64	332	22	272	598	176	2,990	47
159	?	40	171	42	427	380	110	1,710	43
160	7·2	62	566	20	261	262	92	3,400	55
163	6·6	54	300	23	—	500	121	2,250	42
165	?	39	462	17	370	276	82	1,850	48
166	6	55	225	17	—	450	154	2,700	49
167	7	44	204	20	242	380	100	1,940	44
169	9·3	70	342	20	492	244	107	3,590	51
175	6·7	66	350	20	500	350	113	3,500	53
176	7·7	62	378	16	220	379	98	2,650	46

RATIO LIST—Fresh Fœtuses—*continued.*

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
177	7·1	63	501	16	501	286	72	3,010	48
179	8·1	85	264	18	642	529	118	4,500	53
183	6·7	44	542	27	542	380	88	1,900	43
187	5·5	27	101	16	371	171	47	1,115	42
188	8·8	52	142	23	259	1,570	142	2,200	42
191	12	66	225	21	225	307	109	3,380	51
194	8	54	288	34	371	346	130	2,600	48
195	7·1	42	218	20	318	500	106	1,750	42
196	9·3	54	742	29	274	400	153	2,600	48
200	?	36	480	21	360	320	70	1,440	40
203	?	67	335	20	268	319	172	3,350	50
204	9·5	51	518	22	172	235	102	2,590	51
205	?	48	225	26	375	300	83	2,250	47
206	?	54	176	26	331	530	120	2,650	49
207	?	49	414	23	207	518	153	2,070	42
208	8·6	58	236	22	311	369	120	2,950	51
209	?	69	378	30	309	755	139	3,400	49
212	?	45	420	19	466	1,400	196	2,100	47
216	?	48	209	19	306	383	105	2,300	48
218	9·7	50	235	17	391	427	134	2,350	47
220	7·5	85	362	24	290	580	140	4,350	51
225	5·8	41	720	36	514	400	129	1,800	44
226	5·5	31	202	22	444	202	69	1,110	36
229	3	28	505	13	34	336	106	1,010	36
231	9·8	68	356	21	339	475	121	3,560	52
232	7	41	407	15	217	543	776	1,630	40
235	8·9	69	554	23	290	321	145	3,050	44
236	11·2	64	294	22	356	271	133	3,385	53
237	6·6	46	268	22	220	488	92	2,200	48
245	7·5	50	500	24	192	385	156	2,500	50
246	5·8	52	620	17	48	482	79	2,170	42
248	?	68	257	21	300	554	195	3,600	53
252	?	46	274	19	315	315	68	2,050	45
255	9·3	48	717	22	358	331	116	2,150	45
258	?	—	325	18	216	382	105	3,250	—
259	?	55	299	20	359	283	68	2,690	49
261	8·3	56	250	22	323	393	102	2,750	49
263	5·3	49	228	18	300	300	104	2,400	49
264	9	59	299	26	299	352	142	2,990	51
265	8·8	56	151	19	294	407	136	2,650	47
268	?	47	226	25	348	216	89	2,265	48
269	10·2	71	493	25	569	322	130	3,700	52
272	6·1	51	371	22	399	348	143	2,780	54
277	?	49	265	21	265	265	101	2,120	43
283	?	51	466	20	466	509	127	2,800	55
286	?	39	437	22	269	350	140	1,750	45
288	?	83	243	23	370	519	181	3,890	47
293	9·1	65	—	?	—	?	—	3,050	47
294	6·3	49	217	19	181	334	94	2,170	44
295	5·1	31	96	12	192	299	64	1,345	44
296	5·4	30	200	15	218	267	126	1,200	40
298	?	63	826	19	386	252	120	2,894	46

RATIO LIST—Fresh Fœtuses—*continued.*

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
301	7·5	69	308	23	416	644	131	3,540	51
302	7·5	68	181	24	314	363	192	3,450	51
303	?	67	327	25	468	364	173	3,275	49
305	?	54	860	15	215	492	198	2,580	48
311	6·3	76	401	21	472	501	149	4,010	53
312	8	65	363	18	436	344	156	3,270	50
314	8·5	71	213	16	376	337	130	3,200	45
315	7·2	77	370	17	370	352	224	3,700	48
317	7·7	34	364	18	510	236	128	1,275	37
325	?	58	463	20	251	301	134	3,010	52
326	?	67	—	24	455	312	102	2,960	44
331	10·8	83	405	20	258	447	129	4,250	51
334	?	46	1,210	?	457	343	152	2,058	45
337	?	39	270	24	463	405	162	1,620	42
338	8·4	58	538	24	448	598	100	2,690	46
339	?	86	183	17	337	213	109	4,380	51
340	?	79	185	19	335	434	123	3,690	47
341	7·6	68	203	25	339	407	130	3,050	45
343	7·9	53	144	21	288	359	123	2,159	41
344	9·9	57	173	22	289	334	128	2,170	38
346	?	67	240	14	240	360	118	3,600	54
347	?	46	206	18	217	289	103	2,170	47
349	9·1	62	172	22	440	355	92	3,370	54
350	7·8	68	222	15	178	268	111	3,220	47
352	?	45	206	16	275	412	100	2,060	46
353	9·5	73	618	16	293	412	122	3,710	51
355	?	57	146	16	173	206	91	2,780	49
356	?	63	222	14	394	338	127	3,550	56
358	?	61	408	26	341	384	112	3,070	50
360	5·2	47	216	15	288	360	100	2,160	46
361	?	55	232	15	257	467	132	2,570	47
363	?	59	244	15	234	254	133	3,050	52
364	?	58	370	17	370	272	90	2,590	45
365	1·8	31	2,200	20	122	177	92	1,100	36
366	?	—	204	23	275	583	121	2,450	—
368	6·5	—	223	24	220	410	63	2,460	—
369	9	64	315	25	425	375	160	3,150	49
372	10·3	49	415	21	230	519	152	2,075	42
373	11	41	275	22	394	215	90	1,870	48
374	9	74	307	20	307	209	84	3,690	50
379	9·2	59	230	21	263	369	108	2,767	47
381	?	59	495	19	292	264	166	3,070	52
383	4·8	30	331	26	773	178	66	1,160	39
386	?	60	520	15	520	368	74	3,120	52
389	6·8	65	307	17	323	384	133	3,070	47
390	6·5	36	226	20	588	283	95	1.470	41
393	?	29	297	22	576	281	84	1,040	36
395	?	64	3,371	16	590	311	169	2,360	37
397	?	58	268	16	494	257	121	3,215	54
398	?	53	307	22	314	335	157	2,670	50
400	?	58	274	18	287	430	73	3,010	52
401	5·8	57	242	18	318	344	101	3,025	53

RATIO LIST—Fresh Fœtuses—*continued.*

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
404A	10·6	57	282	18	620	258	132	3,100	54
404B	10	55	268	18	512	165	94	2,820	51
405	4	32	676	24	460	288	94	1,150	36
406	6	52	167	15	223	259	88	2,670	51
409	?	49	237	17	276	356	138	2,490	51
410	?	66	630	15	420	350	110	3,150	48
411	?	64	650	15	342	309	103	3,250	51
414	?	67	227	20	253	286	106	3,290	49
415	?	59	..	18	438	?	146	3,070	52
417	6·5	59	258	20	230	?	92	3,230	55
418	?	64	—	?	?	?	?	3,203	50
419	7	65	857	27	428	343	82	3,430	53
420	?	55	393	20	550	306	110	2,750	50
421	?	59	—	21	612	612	122	3,061	52
422	?	82	324	14	504	454	181	4,536	55

RATIO LIST.

MACERATED FŒTUSES.

5	8·8	?	1,577	37	207	484	107	3,628	—
7	?	?	?	?	?	?	?	?	—
11	7·4	37	?	?	?	?	?	2,010	54
18	5·1	75	388	50	466	539	192	3,451	46
22	?	46	?	43	548	—	..	1,920	42
25	7·5	29	756	44	515	454	?	1,134	39
26	6·6	41	?	?	?	?	?	1,728	42
30	12·9	?	638	73	829	1,037	207	4,149	—
32	11·2	?	466	37	866	606	168	3,032	—
34	10·9	?	749	40	238	874	114	2,621	—
38	8·9	45	1,467	34	453	?	162	2,494	55
44	5·5	23	?	54	323	?	176	970	42
49	?	40	708	34	442	737	208	1,771	44
60	7·4	56	375	37	327	712	208	2,850	51
68	3·2	39	455	50	260	607	128	1,820	47
80	?	28	?	38	?	?	128	1,190	42
104	?	26	?	?	?	?	?	930	36
105	?	38	1,678	16	157	244	215	1,510	40
110	5·3	29	585	24	212	585	82	1,170	40
115	?	29	1,155	33	693	297	93	1,040	36
122	?	45	341	76	554	820	277	2,050	46
127	9·5	58	722	38	352	642	171	2,891	50
133	8·3	56	333	30	500	305	147	2,500	45
150	9·5	63	?	46	1,475	656	347	2,950	47
152	4·4	33	866	12	185	289	72	1,300	40
164	?	29	416	6	346	316	182	1,040	36
168	5·5	50	255	27	283	945	119	2,550	51
170	8·7	69	231	26	513	561	211	3,590	52
180	10·8	72	633	27	543	345	122	3,800	53
184	11	63	400	29	426	533	128	3,200	51

RATIO LIST—Macerated Fœtuses—*continued.*

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
185	10·5	39	483	27	845	376	121	1,690	43
189	?	30	?	36	590	236	103	1,180	40
190	3·8	65	?	18	133	443	111	2,660	41
192	8·6	56	416	25	333	500	116	2,500	45
197	7·8	62	420	21	210	394	210	3,150	49
198	9·1	39	1,640	54	820	410	131	1,640	42
201	6·5	49	686	20	369	436	72	2,400	49
210	3·8	27	373	56	448	448	187	1,120	41
211	7·9	66	540	40	263	540	180	2,700	41
215	7·8	54	303	47	344	860	156	2,580	48
217	8·9	26	713	48	713	535	119	1,070	41
219	?	43	775	45	516	517	124	1,550	36
221	6·2	46	440	30	628	880	119	2,200	48
224	?	35	785	70	523	523	185	1,570	45
228	8·9	53	500	21	714	385	147	2,500	47
230	9·4	78	1,157	75	?	900	180	4.050	52
234	?	52	628	14	64	232	98	2,200	42
238	9·5	89	887	24	542	751	264	4,880	55
239	?	63	800	22	457	355	136	3,200	51
247	?	57	295	17	165	460	173	2,070	36
251	9·7	45	1,167	38	388	318	195	1,750	39
257	?	25	590	44	885	885	177	885	36
262	?	38	1,200	58	514	450	156	1,800	48
267	?	53	360	29	317	360	87	2,700	51
270	?	25	416	61	693	260	122	1,040	42
271	?	47	482	19	301	321	134	2,410	51
273	13·5	90	918	36	655	706	195	4,590	51
275	6·7	49	420	32	323	600	150	2,100	43
278	11·8	63	295	34	368	655	190	2,950	47
279	?	53	836	36	418	557	100	2,508	47
280	4·5	20	1,560	52	390	260	120	780	39
281	3·7	26	280	25	490	306	109	980	38
282	5·5	50	281	30	301	?	?	2,110	42
287	4·5	32	675	28	450	338	180	1,350	42
290	5	48	369	17	206	253	91	2,585	54
291	3·3	35	645	23	586	586	143	1,290	37
292	5·4	50	720	51	540	480	309	2,160	45
297	5·6	24	1,518	47	759	379	117	759	32
308	7·5	59	257	40	415	386	164	2,700	46
309	7·6	69	346	18	330	398	115	3,460	50
313	7·5	64	963	19	289	525	128	2,890	45
319	6·6	45	354	19	354	250	72	2,126	47
321	?	28	525	44	700	420	123	1,050	37
322	10·7	50	671	47	470	470	138	2,350	47
323	12·5	49	607	62	850	606	304	2,125	43
335	4·3	47	341	19	455	410	100	2.050	43
336	?	32	673	48	505	673	202	1.010	35
348	6·7	31	1,080	43	635	432	161	1,080	35
357	8·9	50	828	58	824	690	111	2,070	41
359	8	54	1,400	67	840	600	183	2,100	39
362	?	69	377	21	290	191	74	3,770	55
367	7·9		222	24	473	577	97	1,270	—

RATIO LIST—Macerated Fœtuses—*continued.*

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
370	8·6	47	315	19	456	455	100	2,050	44
371	13·3	74	500	25	576	326	132	3,750	51
376	4·2	30	900	35	450	422	155	1,350	45
382	?	35	289	17	636	318	82	1,590	45
384	?	55	481	20	265	212	66	2,650	48
385	?	35	866	52	371	520	144	1,300	37
387	12·3	48	780	45	585	390	104	2,340	47
388	?	44	384	25	602	422	242	2,110	48
392	?	48	1,000	20	714	385	106	2,500	52
394	7·1	30	758	75	559	228	139	1,138	38
403A	?	51	346	23	302	390	167	2,420	47
403B	?	49	357	21	294	238	130	2,500	51
407	7·2	56	331	21	551	248	101	2,480	44
412	7·2	60	347	36	?	480	240	3,120	52

RATIO LIST.

SYPHILIS.

4	5·5	?	277	24	139	?	138	1,940	—
12	5	37	357	26	572	298	95	1,430	39
14	5·5	52	538	13	124	288	78	1,990	38
21	3·3	36	1,090	12	62	321	176	1,090	30
29	5·2	?	588	18	163	490	89	1,470	—
31	3·1	?	615	16	177	369	84	1,474	—
40	3	23	615	26	228	533	100	800	35
41	7·3	55	270	22	263	300	115	2,704	49
53	3·8	17	416	19	119	329	87	625	36
61	3·1	24	286	14	71	161	59	1,000	42
62	7·8	56	294	18	250	416	125	2,500	45
65	3	49	780	26	278	300	150	1,950	40
79	7	44	388	23	125	389	102	1.750	40
84	?	66	440	9	55	509	103	3,300	50
102	6·1	27	525	25	350	169	91	1,050	39
109	4·8	30	1,100	7	110	478	73	1,100	37
118	5·6	35	?	14	131	208	77	1,250	36
124	9·8	53	463	30	510	567	113	2,550	48
129	7	34	388	24	119	517	122	1,550	46
132	10	33	950	25	200	345	127	1,400	43
141	4	21	?	22	187	250	117	750	35
148	6·7	27	208	33	297	416	173	1,040	39
149	6·5	35	289	29	590	433	115	1,300	37
176	7·5	62	378	16	220	379	98	2,650	46
181	7·6	58	667	30	370	445	52	2,670	46
186	?	26	300	12	141	240	60	1,200	47
193	5·4	56	750	26	125	409	129	2,250	40
222	?	46	?	27	171	514	120	1,800	39
227	2·8	18	590	28	184	197	98	590	33

RATIO LIST—Syphilis—*continued.*

No.	Placenta.	Length.	Thymus.	Liver.	Spleen.	Suprarenals.	Kidneys.	Weight of Fœtus.	Length of Fœtus.
229	3	28	505	13	35	336	106	1,010	36
243	?	34	546	25	105	546	94	1,365	40
246	6	52	620	17	48	482	79	2,170	42
328	3·3	24	322	9	132	205	61	1,125	46
332	3·8	49	571	13	97	364	129	2,000	41
342	11·1	56	502	23	89	314	119	2,510	45
377	5	43	1,010	12	72	269	71	2,020	47
391	?	31	354	17	124	191	85	1,240	40
396	?	55	110	25	201	456	149	2,420	44
399	?	34	872	20	106	394	170	1,220	36
413	3·9	28	183	21	275	407	156	1,100	39
416	3·7	45	921	19	91	?	74	1,842	41

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Fig. 1. (Case 420.)

Incomplete tear of Tentorium Cerebelli on right side; the tear is across the fibres at the base of the anterior vertical band. The head shows extreme moulding after labour with generally contracted pelvis. Note elevation of apex of Tentorium and change in direction of vein of Galen and straight sinus.

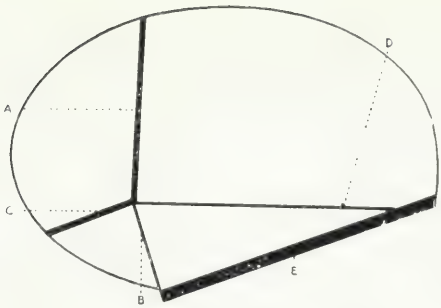

FIG. 2 I.

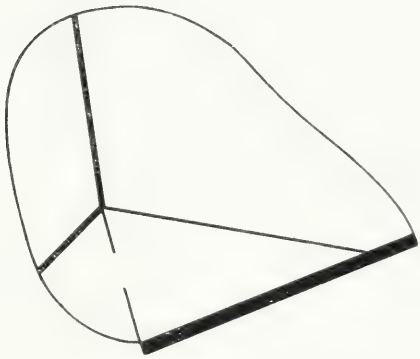

FIG. 2 –II.

FIG. 2 - III.

FIG. 2 I, II, III.

Diagrammatic outlines of head and septal system of dura mater. The lines representing the septa show their chief directions of strength (A) Falx cerebri, (B) middle (vertical) part of Tentorium Cerebelli, (C) line of junction of Tentorium and Falx, (D) anterior (horizontal) part of Tentorium Cerebelli, (E) base of skull.—I. Normal shape of head. II. Shape of head during compression in occipito-frontal diameter; greatest strain on vertical part of Tentorium. III. Shape of head during compression between vault and base; greatest strain on horizontal part of Tentorium.

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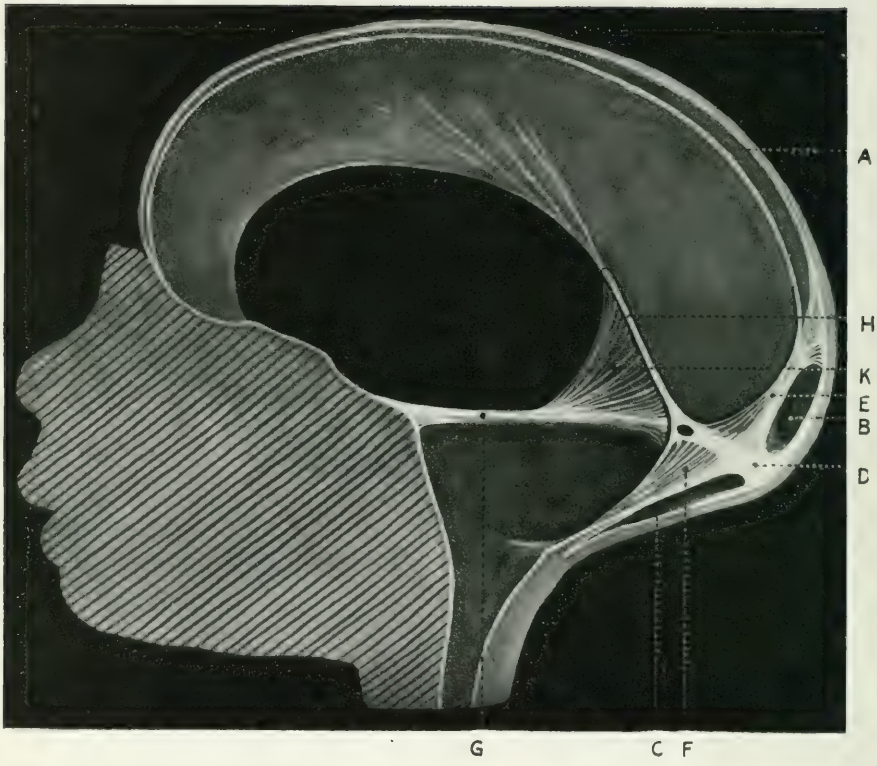

FIG. 3.

Semi-diagrammatic drawing to show arrangement of strengthening bands of antero-posterior system. The head has been bisected sagitally immediately to the left of the Flax Cerebri.—(A) points to superior longitudinal sinus; (B) to Torcular Herophili; (C) to occipital sinus; (D) to main mass of fibrous tissue; (E) to band descending from posterior end of superior longitudinal sinus; (F) to Falx Cerebelli; (G) to deep horizontal band; (H) to white line; (K) to triangular area in anterior part of Tentorium.

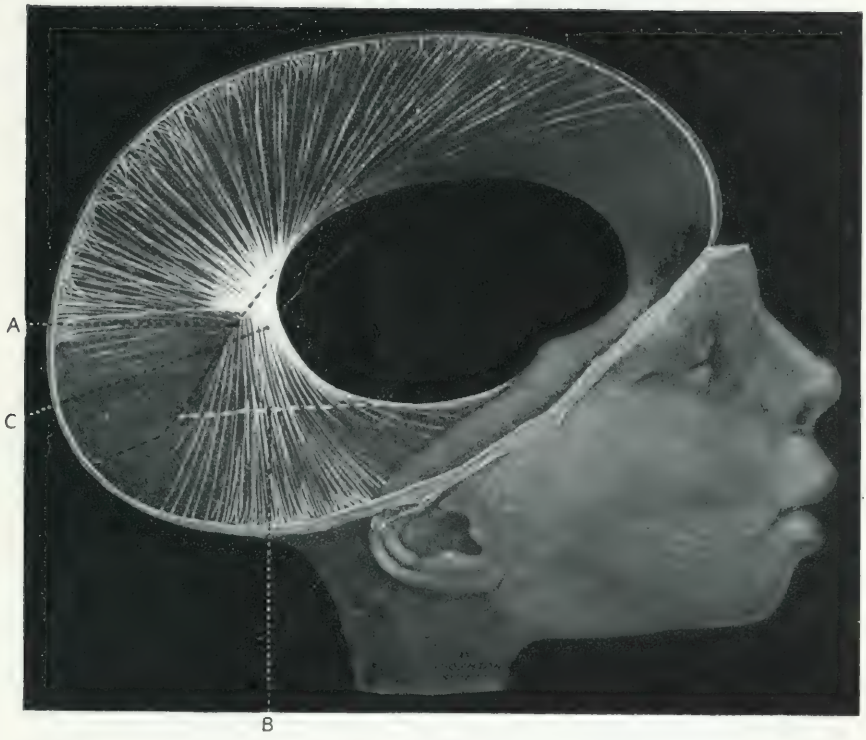

Fig. 4.

Semi-diagrammatic drawing to show arrangement of fibres in Falx and in superficial layer of Tentorium, after removal of parietal and part of occipital and frontal bones.—(A) points to black dotted line indicating floor of straight sinus; (B) points to white dotted line indicating position of deep horizontal band; (C) points to anterior vertical band.

Fig. 8 (Case 340).

Complete tear of Tentorium Cerebelli on right side. The tear is a large perforation and involves all the superficial strengthening fibres of the Tentorium and the base of the deep horizontal band.

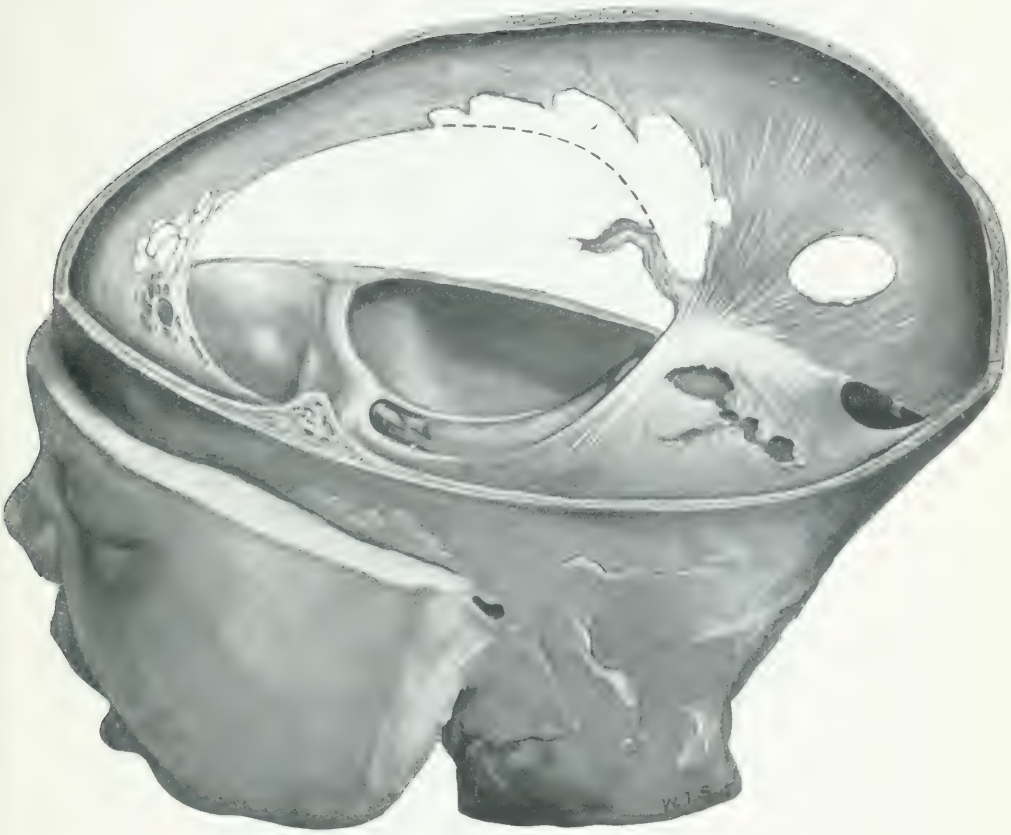

Fig. 9 (Case 220).

Complete tears of Tentorium and extensive tearing of Falx. In the Tentorium there are two perforating tears on the left side; one is a ragged tear starting below the base of the anterior vertical band and extending outwards almost to the lateral border of the Tentorium. The other is a large perforation through the superior wall of the left lateral sinus at its termination in the Torcular Herophili.

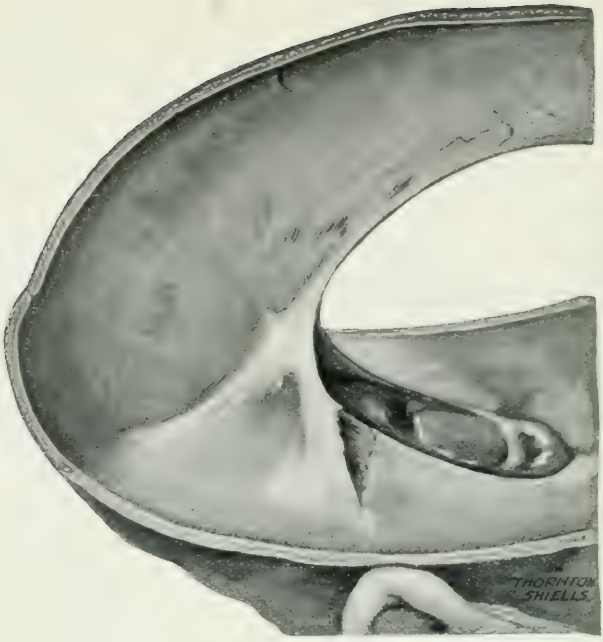

FIG. 10 (Case 265).

Complete tear of Tentorium Cerebelli with separation between superficial and deep layers. The anterior vertical band is widely separated from the deep horizontal band.

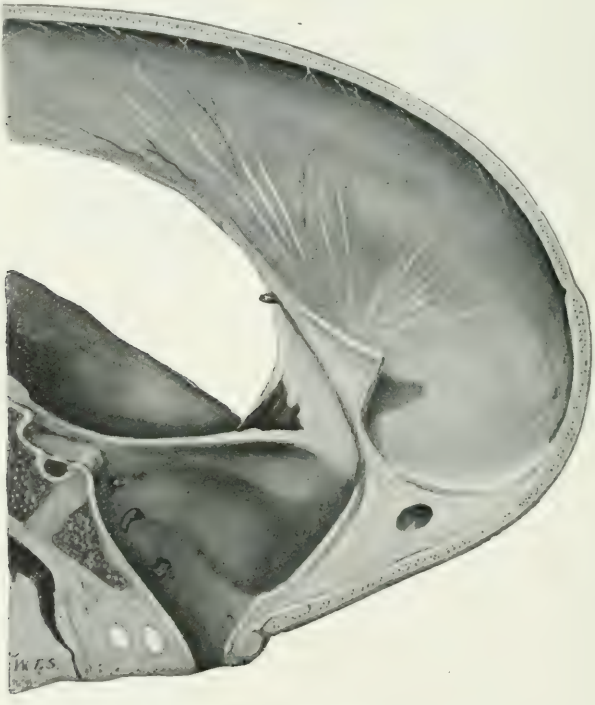

FIG. 11 (Case 265).

Same specimen as in Fig. 10, seen from within after sagittal bisection of head just to left of Falx.

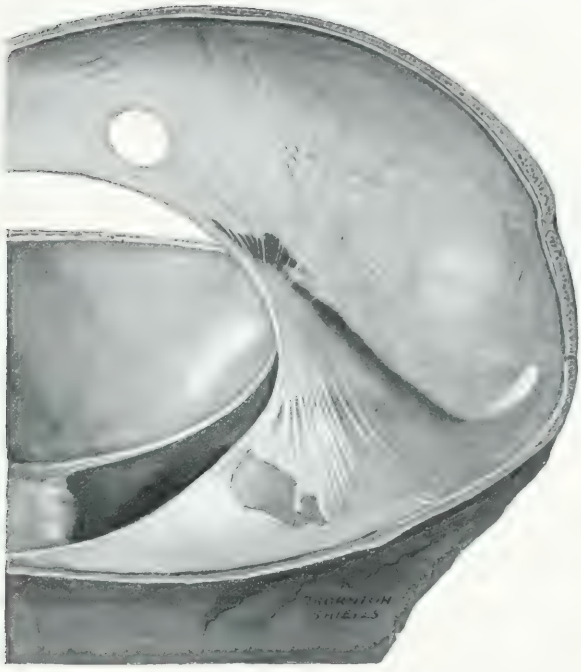

FIG. 12 (Case 179)

Incomplete tear of Tentorium Cerebelli, left side. The superficial fibres have been torn through just below base of anterior vertical band. There is also a small elliptical tear in the Falx

Fig. 13. (Case 140.)

The specimen shows extensive interstitial hæmorrhage or hæmatoma between the layers of the Falx and Tentorium. There are no tears.

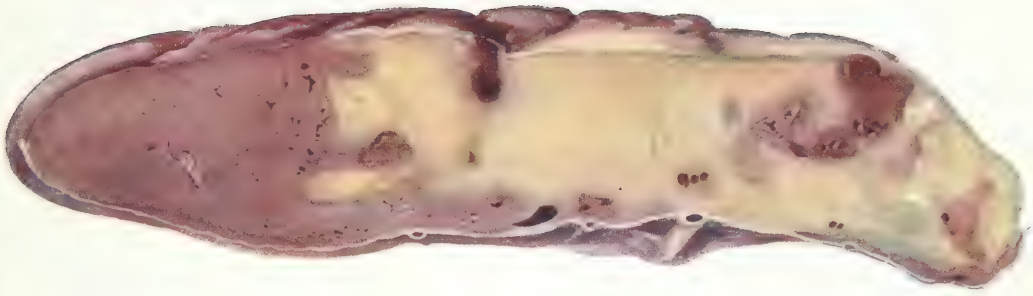

Fig. 14. (Case 225.)

Placenta with very extensive white infarction, occupying two-thirds of its volume (Case 225).

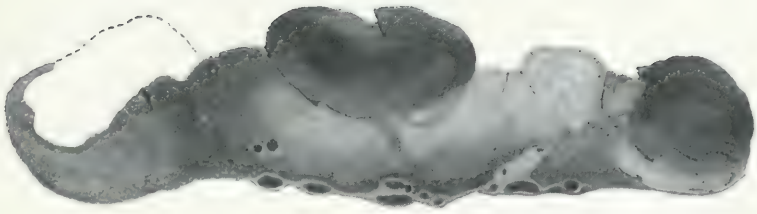

Fig. 15.

Placenta with two retroplacental hæmatomata and red infarction (dark area); from a case of Accidental Hæmorrhage (Case 277).

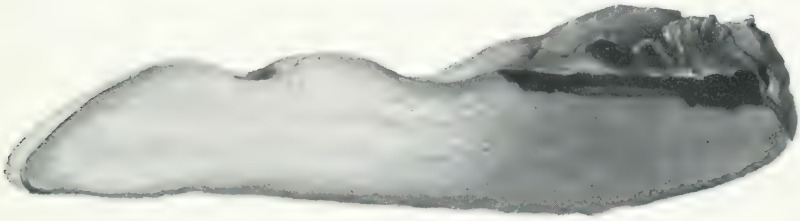

Fig. 16.

Placenta showing an area of red infarction (dark area) subtending a retroplacental hæmorrhage; from a case of Accidental Hæmorrhage with macerated fœtus (Case 335).

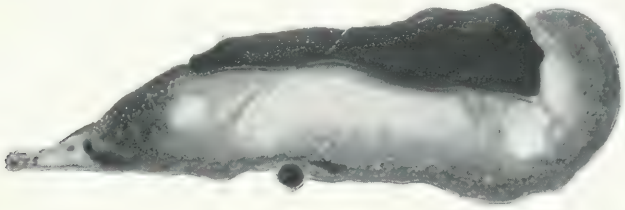

Fig. 17.

Placenta with a large retroplacental hæmatoma (Case 357).

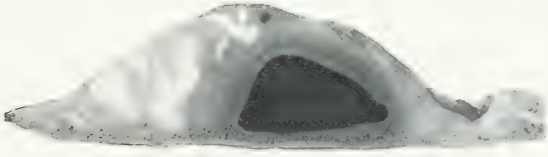

Fig. 18.

Placenta with an intraplacental hæmorrhage (Case 217).

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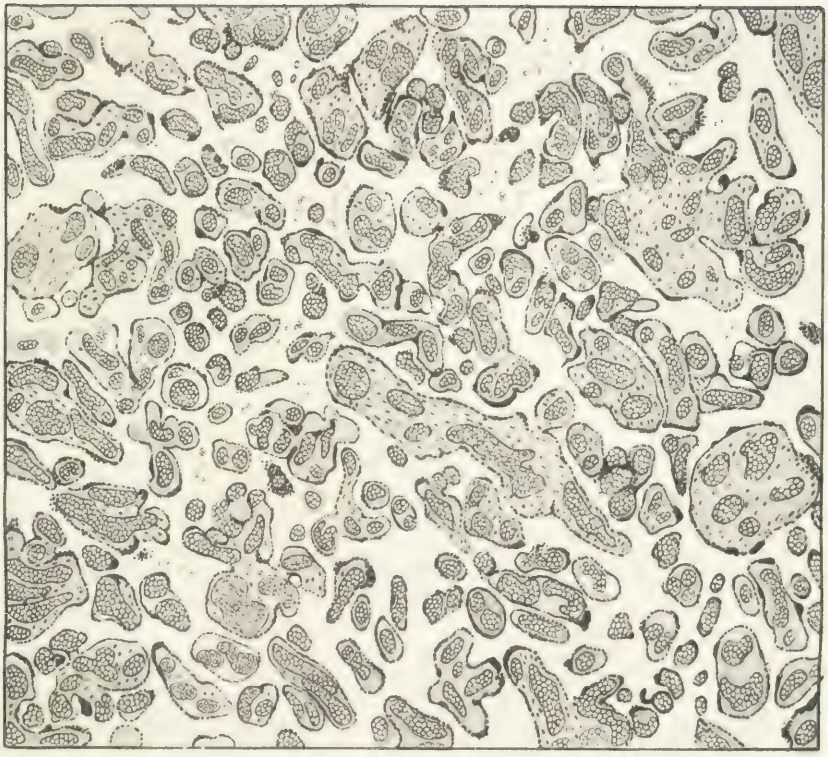

FIG. 19.
Normal placenta: for contrast with Fig. 20.

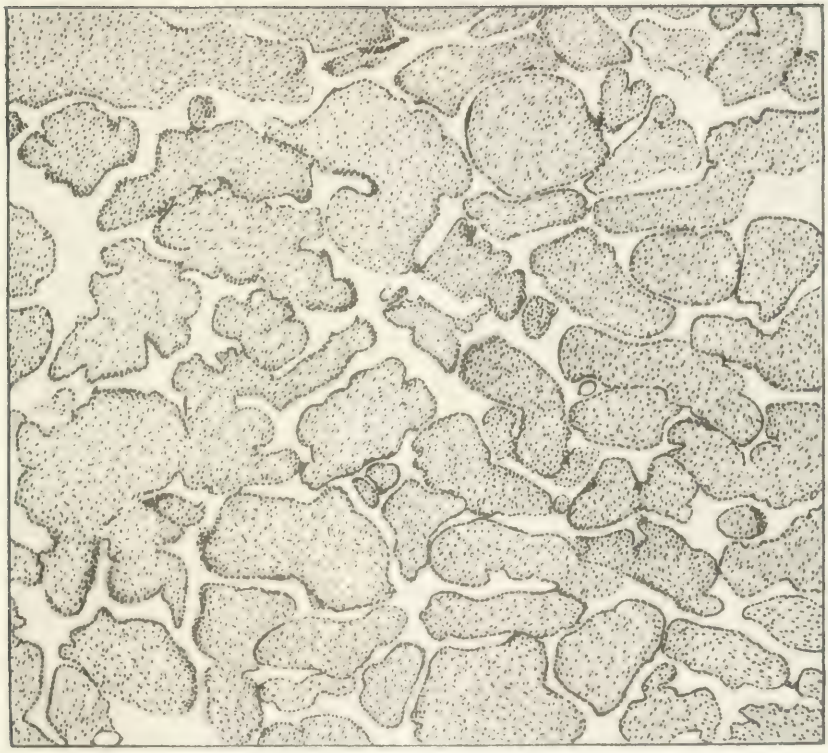

FIG. 20.
Syphilitic placenta.

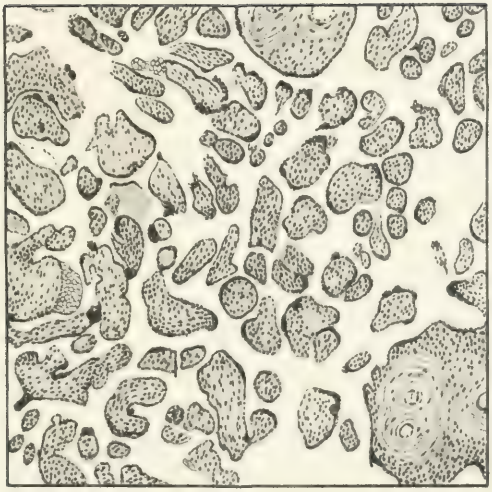

FIG. 21.
A section "suspicious" of syphilis.

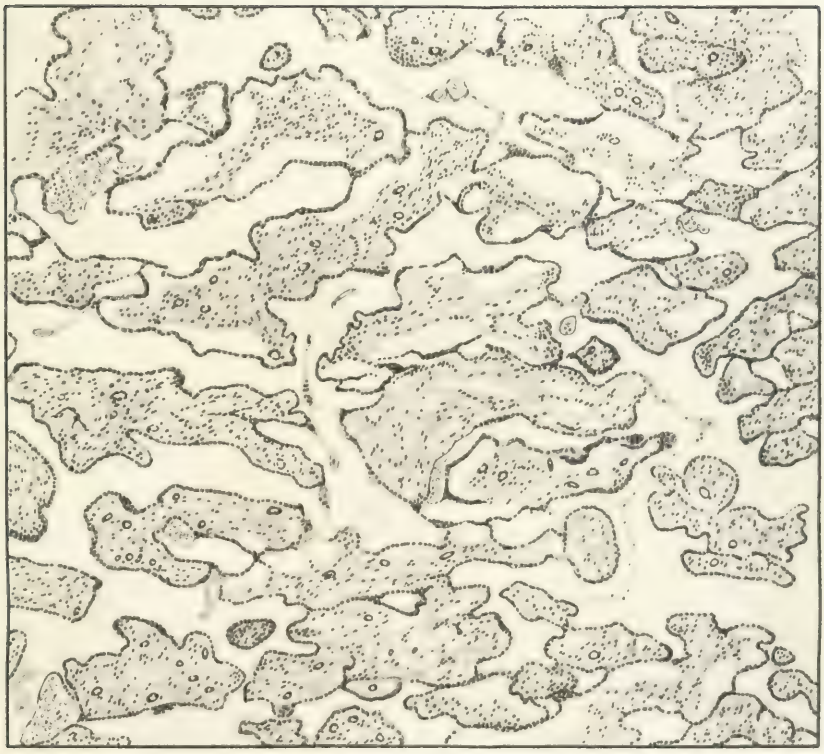

FIG. 22.

Section of placenta from a case of general œdema of the fœtus: the villi are enlarged but are not dense and avascular: the stroma is loose and œdematous. Contrast with Fig. 20.

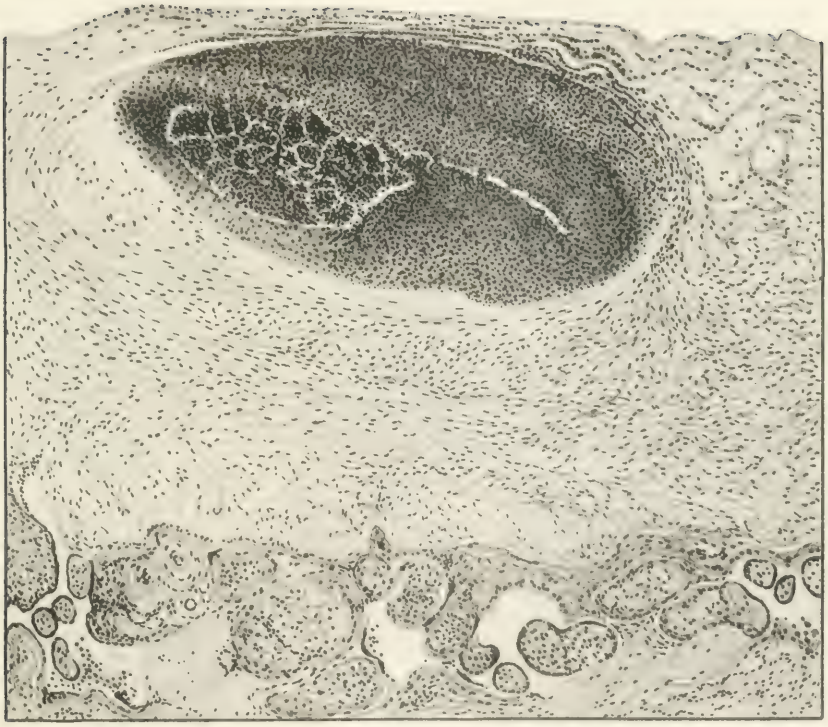

FIG. 23.

Small node of leucocytes in chorionic membrane: probably a gumma.

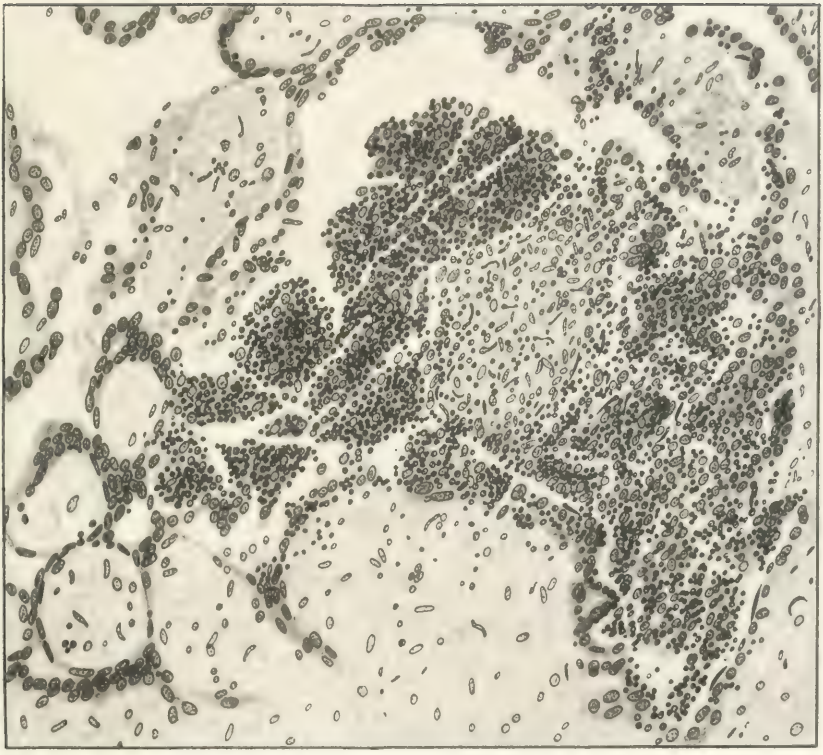

FIG. 24.

Small node of leucocytes at the edge of the decidua probably a gumma.

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